Rapid Review of Clinical ECG

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Second Edition

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Rapid Review of Clinical

ECG

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PREFACE

The Rapid review of practical ECG second edition, maintains its original basic principles to serve medical students and practitioners to provide them a practical insight to rapid comprehensive review in electrocardiography. The new edition is updated and reinforced by ECG papers from our general practice and from my generous colleagues that extensively reviewed and correlated with relevant well-known international references. The current appropriate ACC/AHA/ESC guidelines have also been added. Certainly, there are hundreds of available books in this field, but practitioners, learners, and instructors, need more than enough time to go through these for practical purposes. Therefore, this book is compiled from diverse trustworthy resources to provide an easy reliable approach that can guide systematic and accurate interpretation of ECG paper. My advice to concerned readers is not to confine themselves only to theoretical learning, but also to designate further time for practice and close monitoring relations with patients.

For this edition, we have removed the glossary and counterbalanced throughout the text.

I wish to thank all my colleagues who contributed to this and previous edition, particularly my assistant team. Dr. Ebrahim S. AL-Gubani, Dr. Waleed Q. AL-Awdhaly, Dr. Yousef S. AL-Gubani, and the computer designer Hamed M. AL-Selah.

I am very grateful to my colleagues who extended their help and support, with ECG papers including Al-thawrah hospital.

I would like to thank Dr. S. R. Sharma who carefully read the final press copy.

Saleh M. AL-awdhaly

Dedication and Acknowledgment

To those who strive for learning, writing, and look for every new in medicine and spend their time for the benefit of others.

ABBREVIATIONS

AF	Atrial Fibrillation.
AIVR	Accelerated Idioventricular rhythm
APB	Atrial Premature Beat.
AV	Atrioventricular.
AVNRT	Atrioventricular Nodal re-entery Tachycardia.
AVRT	Atrioventricular re-entry Tachycardia
ASD	Atrial Septal Defect
BBB	Bundle Branch Block.
CAD	Coronary Artery disease.
cm	Centimeter
DCM	Dilated Cardiomyopathy
ECG	Electrocardiography.
HCM	Hypertrophic Cardiomyopathy
HF	Heart Failure
J	Joule
ICD	Implantable Cardioverter Defibrillator
IHD	Ischemic Heart disease.
IVR	Idioventricular rhythm
IV	Intravenous
LA	Left Atrium
LAD	Left Axis Deviation.
LAH	Left Anterior hemi-block.
LBBB	Left Bundle Branch Block.
LPH	Left Posterior hemi-block.
LVH	Left Ventricular Hypertrophy.
MAT	Multifocal Atrial Tachycardia.
MI	Myocardial Infarction.
min	Minute
mm	Millimeter.
msec	Millisecond
mV	Millivolt.
NSR	Normal Sinus Rhythm
NSTEMI	Non-ST Elevation Myocardial infarction
RA	Right Atrium
RAD	Right Axis Deviation.
RBBB	Right Bundle Branch Block.
PSVT	Paroxysmal Supraventricular Tachycardia
RV	Right Ventricle.
RVI	Right atrial infarction
RVH	Right Ventricular Hypertrophy.
Sec	Second
SA	Sino-Atrial.
STEMI	ST Elevation myocardial infarction
SVT	Supraventricular Tachycardia
VF	Ventricular Fibrillation.
VPBs	Ventricular Premature Beats
VSD	Ventricular Septal Defect
VT	Ventricular Tachycardia.
UA	Unstable Angina
WAP	Wandering Atrial Pacemaker
WPW	Wolf-Parkinson-White.

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CHAPTER 1

Basics of ECG

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Basis of Electrocardiography

Definition The ECG is defined as a graphic recording of electrical activity generated by the heart.

Electrical conduction system of the heart

1. Sinoatrial (SA) node

It is the physiological (natural) pacemaker, it has normal intrinsic rate between 60-100 beats/min, the normal heart rhythm resulting from electrical activation in the SA node, is called "sinus rhythm".

2. Internodal pathways

These pathways conduct the impulses between sinoatrial (SA) node and atrioventricular (AV) node; it contains three tracts Anterior, Middle, and Posterior tract.

3. Atrioventricular (AV) Junction

It is the area where the atria join ventricles, it contains atrioventricular (AV) node that delays the impulses coming from SA node, and bundle of His, which acts as a pacemaker when sinoatrial (SA) node and atria fail to produce the impulses. It has intrinsic rate between 40-60 beats/min.

4. Bundle Branches

It is specialized conductive tissue of the ventricles, divided into right bundle branch, and left bundle branch, the later is subdivided to left anterior fascicle (Anterosuperior) and left posterior fascicle (Posteroinferior).

5. Purkinje system

It is specialized conducting cells distributed in the ventricular myocardium.

Diagram 1-1 and figure 1-1 illustrate the normal conductive pathways of the heart:

Diagram 1-1.

1. Sinoatrial (SA) node
2. Internodal pathways
3. Atrioventricular Junction
   - Atrioventricular (AV) node
   - Bundle of His
4. Bundle Branches
   - Right bundle branch
   - Left bundle branch
     - Left anterosuperior fascicle
     - Left posteroinferior fascicle
5. Purkinje system
6. Ventricular myocardium

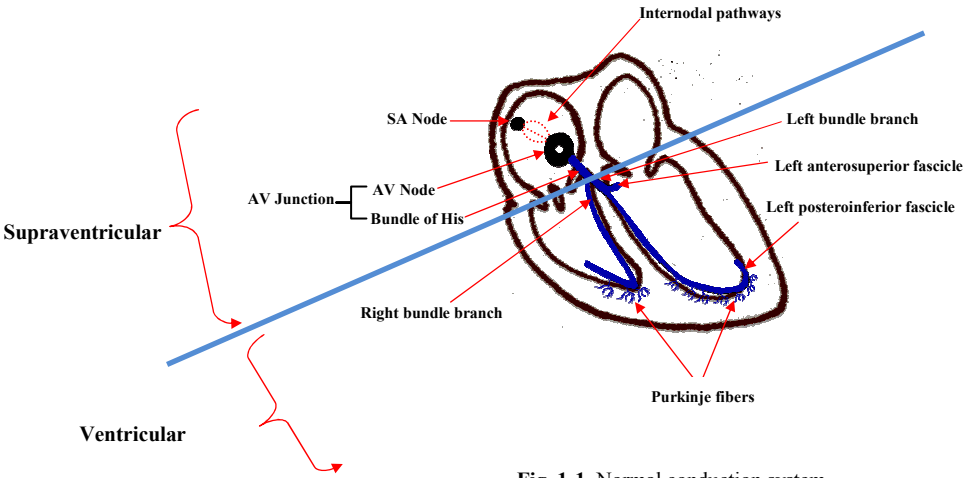


Fig. 1-1. Normal conduction system.

Summary of natural pacemaker sites and their intrinsic rates

1) SA node: 60-100 beats/min, it is the dominant natural pacemaker.
2) Atrial sites: 60-80 beats/min.
3) AV junction: 40-60 beats/min.
4) Ventricular sites: 20-40 beats/min.

Substitute natural pacemaker from which the impulses are released, when the natural pacemaker fails or something interferes with the conduction e.g., block. These impulses called escape rhythm that maintain the electrical activity of the heart at minimum level.

The understanding of the normal conductive system physiology is the key to understand the abnormal rhythm and conduction disturbance, when there is no conduction between atria and ventricles, it is called AV block. The problem inside the ventricular conductive system is called bundle branch block.

The characteristic properties of cardiac tissue:

1) Automaticity is the ability of any cardiac cell to initiate impulses.
2) Conductivity is the ability of cardiac cell to conduct the impulses in sequential manner from above to down under normal circumstances.

Under abnormal circumstances, if the automaticity either increased or decreased, this will result in abnormal cardiac rhythm formation.

If the conductivity is changed either blocked or re-entrant activity appears, then the rhythm changes accordingly.

Normal Sequence of Cardiac Depolarization and Repolarization

The entire cycle that produces the contraction of myocardial tissue can be divided into three phases of electrical activity as follows:

1) During resting state (polarized state), the cardiac muscle has negative charge inside and positive charge outside (Fig. 1-2A).

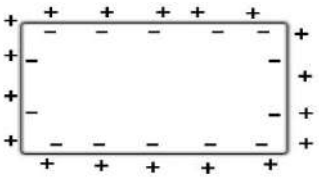


Fig. 1-2 A. Resting state (Polarization)

2) When the cardiac muscle is stimulated by electrical current, its charge becomes reversed, i.e. positive inside, and negative outside. This change is called (depolarization) (Fig. 1-2B).

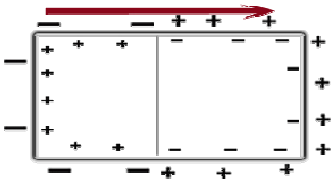


Fig. 1- 2 B. Arrow indicates the direction of depolarization (Excitation)

3) When the wave of excitation is over, the cardiac muscle returns reversely to the original state, i.e., negative inside and positive outside. This process of reversal to resting state is called (repolarization) (Fig. 1-2C).

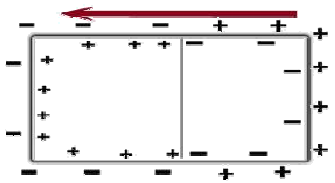


Fig. 1-2 C. Arrow indicates the direction of repolarization (Recovery).

Spread of electrical impulses through the heart

Step 1: atrial depolarization

Impulses initiated in the SA node, activate the atria almost simultaneously by electrical potential. They produce positive upward deflection (P wave) in the leads that lie in the same direction of current flow, e.g., lead II, and negative deflection (inverted P wave) in the leads that lie in the opposite direction of current flow, e.g., lead aVR (Fig. 1-3A).

Fig. 1-3 A. Spread of the impulse from the SA node to the atria

Step 2: AV node depolarization

The impulses pass slowly through the AV node, thus the ventricular depolarization is delayed. This delay appears as an isoelectric segment from the end of the P wave to the starting point of the QRS (known as PR segment) (Fig. 1-3B).

Fig. 1-3 B. Arrow indicates the delay of the impulse in the AV node

Step 3: Septal depolarization

After brief delay of impulses at the AV node, they activate the septum from left to right, this results in small positive deflection (septal r wave) in lead V_1 and small negative deflection (septal q wave) in lead V_6 (Fig. 1-3C).

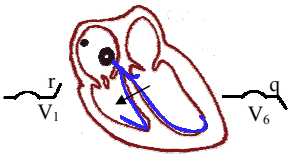


Fig. 1-3 C. Arrow indicates septal depolarization

Step 4: Ventricular depolarization

The ventricular depolarization starts form the endocardium toward the epicardium. As the left ventricle has larger muscle mass than the right ventricle, so, the larger left ventricular force predominates over the smaller right ventricular force. Hence, they cause large positive (upward) deflection (R wave) in the left leads which lie in the same direction of the current flow e.g. V_5 - V_6, and negative deflection (downward) S wave in right leads e.g., V_1, V_2, that lie in opposite direction of current flow (Fig. 1-3 D). The relative increase in the size of the R wave toward the left precordial leads is called normal R wave progression. Lead aVR lies opposite to the direction of the current flow, so the QRS complex is completely negative in this lead.

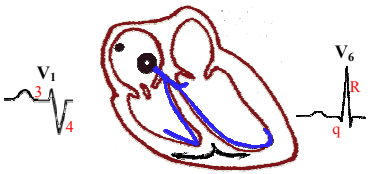
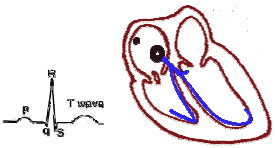


Fig. 1-3 D. Arrows indicate simultaneous ventricular depolarization

Step 5: Ventricular repolarization

When the heart is fully depolarized, there is no electrical activity for a brief period (ST segment). The repolarization starts in the epicardium toward the endocardium, causing upward (positive) deflection (T wave and U wave). The period following T wave until the next P wave with flat baseline is called T-P segment that represents pre-depolarization period (Fig. 1-3E).

Fig. 1-3 E. Notice the ST segment and the T wave reflect electrical current generated during ventricular repolarization



Summary of Spread of electrical impulses in the heart

1) Atrial depolarization represented by the P wave on the ECG paper, that is, positive in lead II and negative in aVR.
2) AV nodal depolarization results in formation of PR segment that is, from the end of the P wave to the beginning of the QRS complex.
3) Septal depolarization normally is from left to right producing small septal r wave in V_1, V_2, and q wave in V_5, V_6.
4) Ventricular depolarization, both ventricles are simultaneously depolarized, resulting in QRS complex.
5) Ventricular repolarization produces ST segment, T, and U waves.

Notes:

- *The small wave of atrial repolarization usually not seen in the ECG, because it is masked by the ventricular depolarization.*
- *Do not confuse between PR segment and PR interval, the latter is the period from the beginning of the P wave to the beginning of the QRS complex.*
- *TP segment is the time from the end of the T wave to the onset of the P wave.*
- *Remember that any lead lies in the direction of the electrical activity has positive deflection, while the lead away from the direction of electrical activity has negative deflection. However, when the electrical activity is perpendicular to any lead, it results in biphasic deflections.*

Shape of different components of QRS complex on ECG paper

- The basic shape of the normal ECG is shown in figure 1-4.

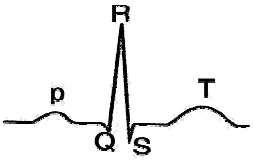


Fig. 1-4. Basic shape of P-QRS-T that formed after complete depolarization-repolarization cycle.

- Knowledge of the different components of the QRS wave is a fundamental step to understand and differentiate the normal and abnormal QRS (Fig. 1-5).

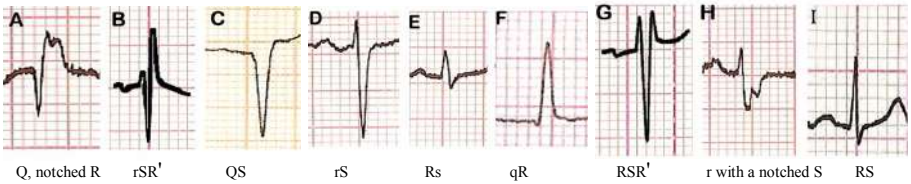


Fig. 1-5. Different shapes of the QRS complex; the waves are termed small q, r, s or capital (larger) letter QRS according to their amplitude (height). Occasionally, the QRS complex contains more than one deflection of the same wave. In such case the extra upward wave is called (R' or r' prime), and the extra downward wave is called (S' or s' prime) shown in. (Fig. 1-5 B and G)

Notes:

The following terms should be kept in mind:

- *Monophasic QRS, present as single upward deflection above the isoelectric line, or downward deflection below the isoelectric line (Fig. 1-5 C).*
- *Biphasic QRS, two deflections, one above and the other below the isoelectric line (positive deflection is equal to negative deflection).*
- *Triphasic QRS, three waves in different directions around the isoelectric line (Fig. 1-5 B and G).*

ECG Paper

1. Horizontal measurement the horizontal measurement represents the time. (i.e., Duration measured in seconds) (Fig. 1-6).

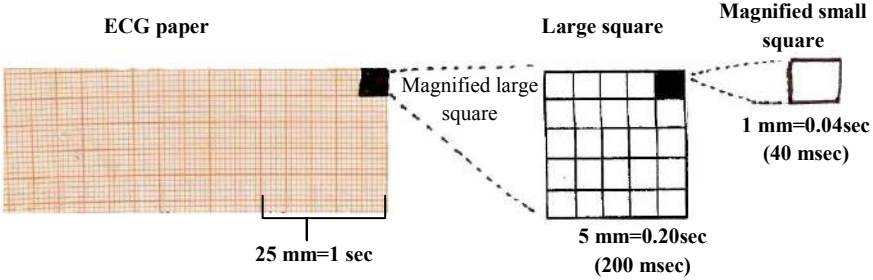


Fig. 1-6.

A| ECG paper is composed of a number of small squares, each small square is equal to 1 mm wide = 0.04 sec = 40 milliseconds.

B| Each 5 small squares (5 mm) wide represent 0.20 sec (i.e., 5× 0.04=0.20 sec) = one large square.

C| Each 5 large squares (25 mm) wide represent 1 sec (i.e., 300 large squares/min, 5× 60=300 or 1500 small squares/min, 25× 60=1500 small squares/min).

D| The ECG paper routinely moves through the machine at constant speed of 25 mm/sec (Fig. 1-7 A and B). The paper speed can be changed to 50 mm/sec (Fig. 1-7 A) or 12.5 mm/sec (Fig. 1-7 B). Any change in the speed of the paper should be noted because it will change the basic pattern of the ECG.

Fig.1-7 A. The usual paper speed 25 mm/sec is changed to the unusual paper speed 50 mm/sec, waves appear abnormally wide and slow.

Fig.1-7 B. The usual paper speed 25 mm/sec is changed to the unusual paper speed 12.5 mm/sec, waves appear abnormally narrow and fast.

2. Vertical measurement

The vertical measurement on ECG paper represents amplitude of a wave extending from the isoelectric baseline to the peak of that wave, measured in millivolt (mV) (Figs. 1-8 A and B).

A| Vertically, the small square (1 mm) represents the amount of electrical potential of 0.1 mV.

B| Each large square (5 mm) represents 0.5 mV.

C| Machine is standardized (calibrated) in such a manner that an impulse of 1 mV will cause deflection of 10 small squares = two large squares.

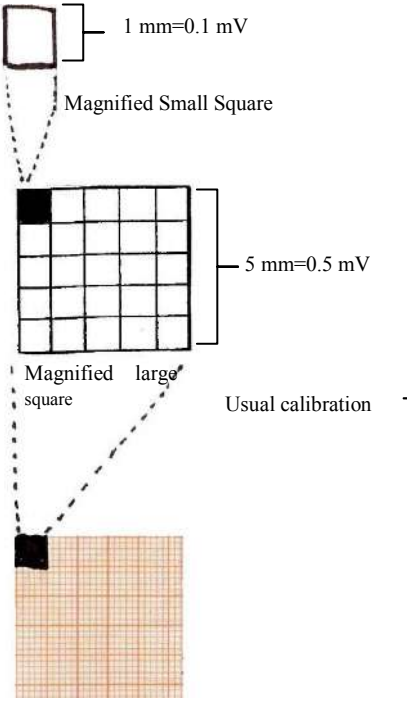


Fig. 1-8 A. ECG paper

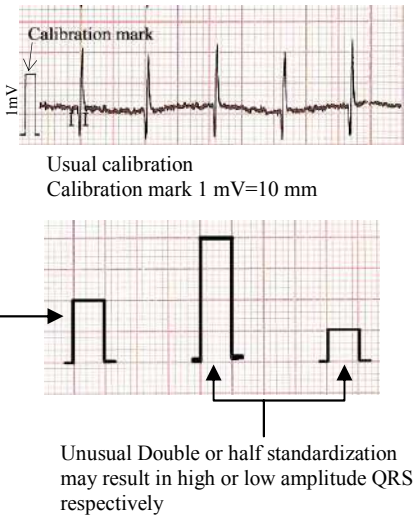


Fig. 1-8 B.

How to calculate the Heart rate

The following methods can be used to calculate the heart rate:

1. When the heart rhythm is regular (sinus rhythm)

A| Count the number of large squares between two consecutive R waves and divide a constant 300 by the number of these large squares.

For example, if there is a QRS complex every two large squares, the heart rate will be 300 ÷ 2 = 150 beats/min (Fig. 1-9A).

- If there are three large squares, the heart rate will be 100/min.

B| Count the number of small squares between two consecutive R waves and divide a constant 1500 by the number of these small squares. In Fig. 1-9A there are 10 small squares between any two consecutive R waves and the heart rate is 1500 ÷ 10=150 beats/min.

Fig.1-9A. Calculation of heart rate when the rhythm is regular.

Look at these equations

$$HR = \frac{300}{x}$$

Where **300** is the constant number, **X** is the number of large squares between two consecutive R waves.

Example Fig. 1-9 A. $\frac{300}{2} = 150$ beats/min

Or the heart rate can be calculated by the number of small squares as follows:

$$HR = \frac{1500}{x}$$

Where **1500** is the constant number, **X** is the number of small squares between two consecutive R waves.

Example Fig. 1-9 A. $\frac{1500}{10} = 150$ beats/min

Practical guideline:

Generally, if you find three to five large squares between two consecutive R waves, the heart rate is within normal range.

2. When the heart rhythm is irregular (the QRS complexes will be at variable distance). So we calculate the heart rate as follows:

A| Count the QRS complexes falling in 30 large squares and multiply it by 10. This will give the heart rate/min (Fig. 1-9B).

Or

B| Count the QRS complexes falling in 15 large squares and multiply it by 20. This will give the heart rate/min.

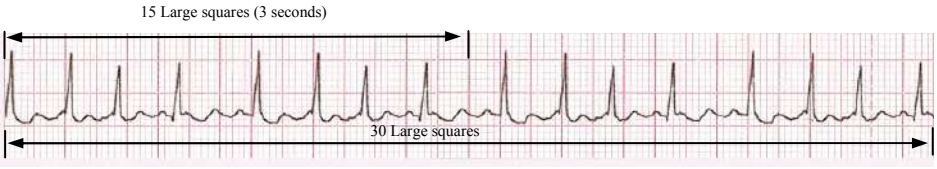


Fig.1-9 B. Calculating heart rate when the rhythm is irregular

Key point:
30 large squares contain 16 QRS complexes.
Therefore, the heart rate 16×10= 160 beats /min.

Other methods (less commonly used in clinical practice)

On the ECG paper, one sec = 5 large squares; therefore, 3 sec = 15 large squares and 6 sec = 30 large squares.

Most recording papers are marked every 15 large squares (3 sec interval) with black mark on the upper border (fig. 1-9C), so we calculate the heart rate as follow:

1) Count the number of QRS per 3 sec interval, and then simply multiply the result by 20 to get the number of beats per minute.

   Or

2) Count the number of QRS per 6 seconds interval then simply multiply the result by 10 to get the number of beats per minute (Fig. 1-9C).

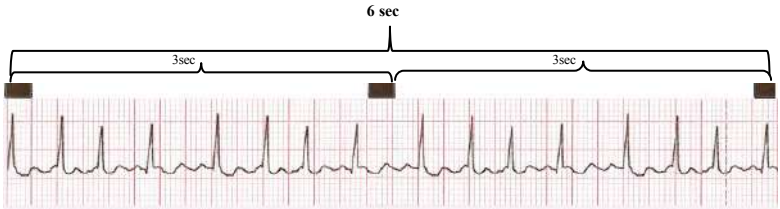


Fig.1-9 C. When the rhythm is irregular, heart rate can be determined by counting the R waves in a 6 sec strip and multiplying by 10 (paper speed is 25 mm/sec).
In this example 6 sec contains 16 QRS complexes so the heart rate is 16×10= 160 beats/min and 3 sec contain 9 QRS complexes so the heart rate is 8×20 = 160 beats/min.

C| The speed of ECG paper is 25 mm/second =2.5 cm/second; therefore, 6 seconds = 2.5× 6 =15 cm, so count the number of QRS complexes in any 15 cm interval, then multiply the result by 10 to get the number of beats/min. We can use a ruler for this calculation if the ECG paper has no marks (Fig. 1-9D).

Fig.1-9 D. Heart rate can be determined by counting the R waves in 15 cm strip and multiplied by 10.
In this example 15 cm contain 16 QRS complexes so the heart rate is 16×10 = 160 beats/min

Electrocardiographic Waves, Intervals, and Segments

The normal ECG waves, intervals, and segments are clarified in the Fig. 1-10 A and B, and summarized in table 1-1.

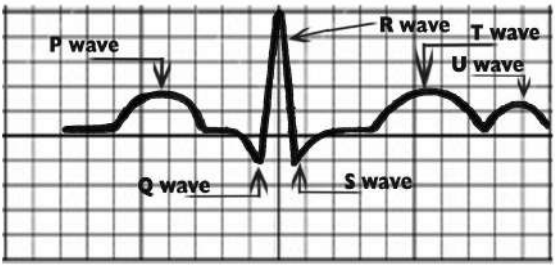


Fig.1-10 A. Waves of the ECG. Though the J point is not labeled in the diagram, it is the junction between the end of the QRS complex and the beginning of the ST segment.

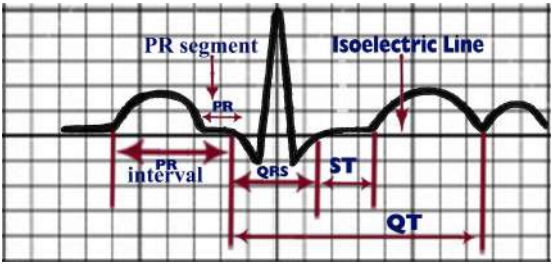


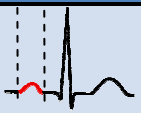
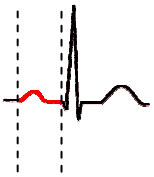
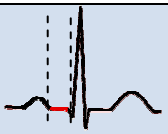
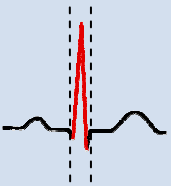
Fig.1-10 B. ECG Intervals and Segments, notice PR segment, and interval have been clarified in the diagram to avoid confusion.

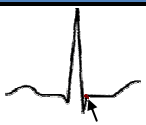
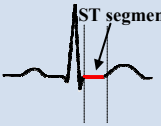
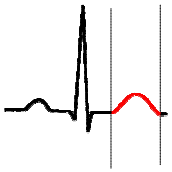
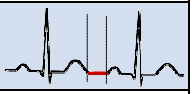
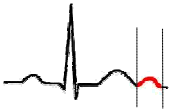
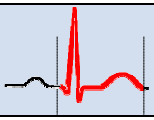
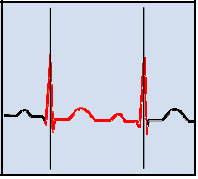
Note:

Keep in mind the recognition of normal paper speed, standardization, correct measurement of waves, intervals, and segments is the key to know abnormal ECG changes.

Isoelectric line: is a flat line on ECG paper, representing neither a positive nor a negative electrical activity ,the recognition of this line is important in clinical practice to differentiate between different types of supraventricular arrhythmias.

Table 1-1. Electrocardiographic Waves, Interval, and Segments

	Duration	Amplitude	Events in the heart	Morphology on ECG paper
P wave	Equal or less than three small squares. 0.12 sec (≤3 mm)	Equal or less than two and one-half small squares (≤2.5 mm)	Atrial depolarization	
PR interval	From the beginning of P wave to the beginning of QRS complex The normal PR interval ranges from 0.12 to 0.20 second (three to five small squares)	PR interval composed of P wave and the isoelectric PR segment	Represents the time interval taken by impulses to pass from the SA node to ventricular muscle	
PR segment	From the end of P wave to the beginning of QRS complex	Isoelectric (flat line)	Delay of the impulse at the AV node	
Q wave	First negative deflection less than one small square (<0.04 sec)	Less than 25% of R wave height in the same lead	Represents the septal activation (recorded in the left precordial leads)	
R wave	First positive deflection of QRS	A- Upper limit for R wave amplitude in leads V_4, V_5 and V_6 =25 mm or may be 30 mm in young adult. B-Lower limit for R wave amplitude in each of leads I, II, and III: 6mm.	Q wave with R wave and S wave form QRS complex which represent ventricular depolarization	
S wave	The second negative deflection follows R wave	A-Upper limit for S wave amplitude in V_1-V_2: 26 mm. B-Lower limit for S wave amplitude in V_1: 3 mm.		
QRS	Beginning of QRS complex to J point where QRS joint ST segment. Less than two and half small squares (<0.10 sec)			

	Duration	Amplitude	Events in the heart	Morphology on ECG paper
J point	**The J point defines the end of the QRS complex and beginning of the ST segment.**	**It is a point**	**End of ventricular depolarization**	 **The J point**
ST segment	**The segment between the end of the QRS complex and the beginning of the T wave**	**Isoelectric (i.e. neither positive nor negative) But may be slightly elevated or depressed.**	**It may represent the pause in ventricular activity before repolarization**	 **ST segment**
T wave	**From the end of the ST segment to the beginning of TP segment**	**1. In limb leads it is nearly less than 6 mm (less than 6 small squares). 2. In chest leads it is also less than 6 mm in women but may be taller in men including in V_2-V_3, but usually not more than 12 mm (12 small squares).**	**Represents the recovery (repolarization) of the myocardium.**	
TP segment	**From the end of T wave to the beginning of the P wave**	**Isoelectric (flat line)**	**Pre-depolarization period**	
U wave	**A wave on the ECG that follows the T wave**	**The U wave is usually absent from most leads of normal ECG, so any prominent U wave is clinically significant**	**Represents the last phase of ventricular repolarization**	
QT interval	**QT interval extends from the beginning of the QRS complex to the end of T wave and not the U wave (if present).**	**It is an interval**	**Represents the total ventricular depolarization and repolarization**	
QTc Corrected QT interval	**With normal heart rate 60-100 beats/min QTc = 0.39 – 0.46 sec Or roughly QTc is less than half of the RR interval**	**It is an Corrected interval**	**Represents the Corrected total ventricular depolarization and repolarization according to the given heart rate**	**It is essential to correct the QT interval according to the given heart rate, correction of QT is done by using Bazett's formula as follows:** $QTc = \frac{QT}{\sqrt{RR}}$
RR interval	**Is the time between two consecutive R waves, it is either measured directly from ECG or calculated by dividing 60 by the patient heart rate. Example at heart rate 80 beats/min the RR interval is 60/80 =0.75 sec**		**It is the time between two consecutive ventricular depolarization**	

Sources; informations included in this table adopted from:

1) Convor, 2003, pp. 14-15.
2) Myers et al (eds), Software on Scheid's Basic ECG, 1996

ECG Leads

The standard ECG is composed of six limb and six chest leads

Limb leads

These leads are recorded by placing electrodes on the right arm, left arm and left leg as follows:

A| **Bipolar (standard) leads (I, II, III)** Called bipolar because it records the electrical potential difference between voltages (from the heart) detected at two extremities (positive "+ve" and negative "–ve" (Fig. 1-11A)).

Lead I the positive electrode is placed on left arm (LA), and the negative electrode on right arm (RA). (I = LA–RA) i.e., it records voltage difference between left arm and right arm.

Lead II this is recorded by placing positive electrode on left Leg (LL), while negative electrode is placed on right arm (RA). (II= LL– RA) i.e., it records voltage difference between left leg and right arm.

Lead III this is recorded by placing the positive electrode on left leg (LL) while the negative electrode is placed on left arm (LA). (III= LL– LA) i.e., records voltage difference between left leg and left arm.

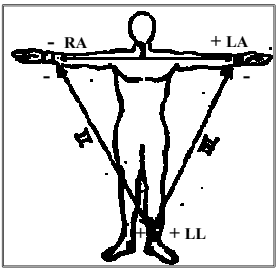


Fig. 1-11A. Bipolar limb leads

The bipolar standard limb leads I, II, and III represented by Einthoven's triangle (Fig. 1-11B)

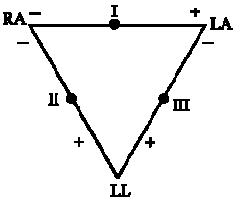


Fig. 1-11 B. Einthoven's Triangle

Einthoven's triangle is redrawn where lead I, II and III intersect at a common central point. The result is the triaxial diagram (Fig. 1-11C), the bipolar leads are related by the following equation:

$$II = III + I$$

$$\text{Lead I} = (\cancel{LA} - RA)$$

$$\text{Lead III} = (LL - \cancel{LA})$$

$$LL - RA = II$$

(i.e., II= I + III)

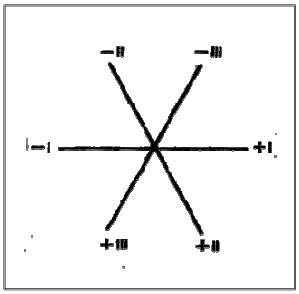


Fig. 1-11 C. The triangle is converted to a triaxial diagram.

Example (Fig. 1-11D)

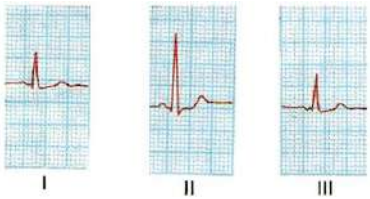


Fig. 1-11 D. ECG example, with normal heart axis.
Lead II = I+ III

B| **Unipolar Leads** Augmented1[1] limb leads aVR, aVL, and aVF

Unipolar Leads is called so, because the centre of the heart is used as a reference point and the positive electrode is placed on the limbs and used as another point (Fig. 1-12A). i.e., it records the electrical voltage at one location related to zero potential, rather than related to voltages at another extremity, as in case of bipolar leads.

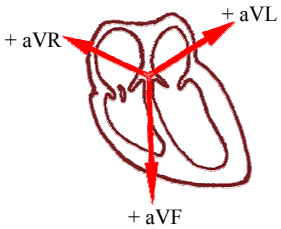


Fig. 1-12 A. The direction of unipolar limb leads in relation to the central point of the heart

Abbreviations aVR, aVL, and aVF stand for:

aVR = augmented voltage right arm.

aVL = augmented voltage left arm.

aVF = augmented voltage foot (left).

In the augmented limb leads, one limb electrode is used as positive pole and the other two are joined to form a ground reference (Fig. 1-12B).

aVR directed from the right arm, which is positive in consideration to other unipolar leads.

aVL directed from the left arm, which is positive in consideration to other unipolar leads.

aVF directed from the left foot, which is positive in consideration to other unipolar leads.

[1] **Augmented**: In order to monitor a lead in this manner, the voltage in the ECG machine is amplified (augmented). This is needed to get tracing of the same magnitude as leads I, II, III. This increases the size of potential by 50% without any change in configuration from the non-augmented record.

The unipolar leads are represented by triangle (Fig. 1-12B).

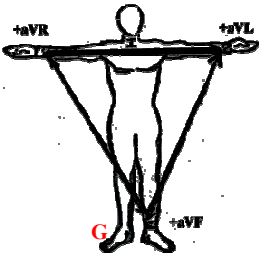


Fig. 1-12 B. Triangle of unipolar limb leads. G indicates position of ground electrode[1]

The triangle is converted to a triaxial diagram (Fig. 1-12C).

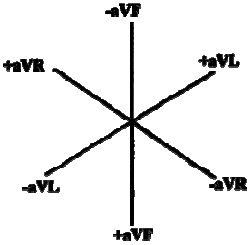
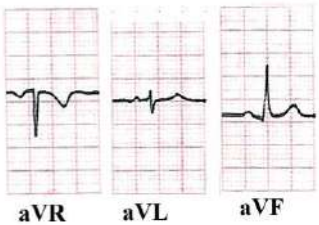


Fig. 1-12 C. Triaxial diagram of unipolar limb leads

The unipolar (augmented) limb leads also related by simple equation

(aVR + aVL + aVF = Zero)

For example (Fig. 1-12D):



aVR = -10 mm

aVL = +3 mm – 3 mm

aVF = + 10 mm

aVR +aVF + aVL = - 10 + (+3-3) + (+10) = 0

Fig. 1-12 D. example, with normal heart axis, the net magnitude of aVR aVL, and aVF = zero

[1] The right leg electrode functions as a ground (earth) to prevent interference of the electrical currents in the system.

Derivation of hexaxial lead diagram

The three unipolar and the three bipolar triaxial leads can be shown on the same diagram, producing (hexaxial leads diagram) (Fig. 1-12 E).

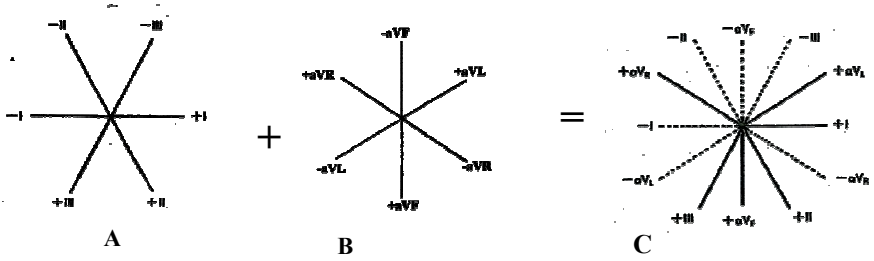


Fig. 1-12 E. reveals the combination of two triaxial references system

A&B resulting in hexaxial references system C.

A| Triaxial diagram of the bipolar leads (I, II, III).
B| Triaxial diagram of the unipolar leads (aVR, aVL, aVF).
C| The two triaxial diagrams can be combined into hexaxial diagram.

Each unipolar limb lead is perpendicular to one of the bipolar limb leads (Fig. 1-12F) as follows:

aVL is perpendicular to lead II.

aVR is perpendicular to lead III.

aVF is perpendicular to lead I.

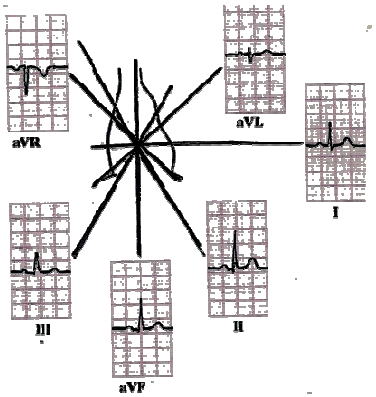


Fig. 1-12 F. The ECG pattern recorded by the six "standard" leads

Chest Leads

Precordial (chest) leads (V_1 to V_6) are obtained by direct recording over the chest itself (Fig. 1-13A).

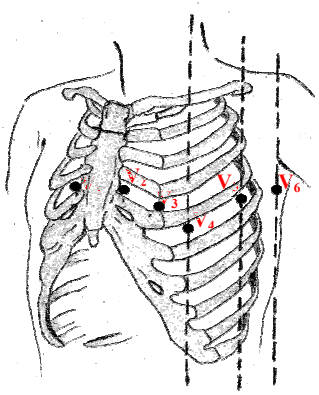


Fig. 1-13 A. The sites of electrode placement on the precordium.

V_1: Fourth intercostal space at right sternal border.

V_2: Fourth intercostal space at left sternal border.

V_3: Midway between V_2 and V_4.

V_4: Midclavicular line at fifth intercostal space.

V_5: The left anterior axillary line at fifth intercostal space.

V_6: The left mid-axillary line at the same level with V_4 and V_5.

Additional chest leads additional leads that may be needed in special situations as follows:

A| Right sided chest leads

The Patients with inferior myocardial infarction should have right sided ECG if right ventricular infarction is suspected, which occurs almost exclusively in the setting of inferior myocardial infarction. See the position of right side chest leads (Fig. 1-13B).

Right ventricular leads V_{2R}, V_{3R}, V_{4R} on the right chest comparable to V_2, V_3, V_4 respectively (Fig. 1-13A).

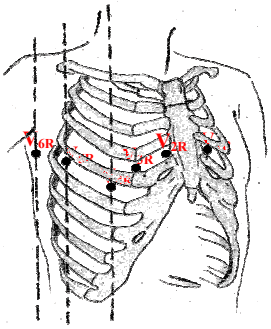


Fig.1-13 B. Drawing shows positions of right sided chest leads

B| Posterior ECG leads

The Patients suspected of posterior myocardial infarction should have posterior ECG leads (V_7 through V_9) (Fig. 1-13 C). ST segments elevation in these leads increase the specificity for diagnosis of posterior MI (regarding reperfusion therapy).

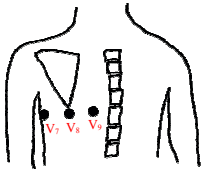


Fig.1-13 C. Drawing shows positions of posterior chest leads

Note:
Because the colors of wires of ECG leads are different between American or European machines, it is important to know about the machine you are using.

Cardiac axis

The cardiac axis is an indicator of general direction of the electrical current through the heart.

The mean QRS axis is the sum of all electrical current through the heart during depolarization, which represents an ECG waveform measured in degrees in frontal plane (Fig. 1-4).

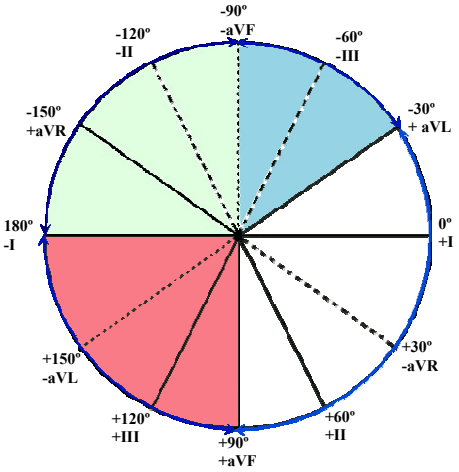


Fig.1-14. Represents hexaxial diagram for frontal plane axis determination.

1) **The Frontal plane axis of the QRS complex** It is the sum of the electrical axis of the QRS complex, which measured in reference to the six limb leads (frontal plane). In general practice it consists of the following:

   A| The Normal axis

   In adults, the normal QRS axis is considered to be within (- 30º and +90º) (Fig. 1-14).

B| Right axis deviation (RAD)

The Right axis deviation in adults is from + 90° to -180° (Figs. 1-14 and 1-15).

Causes of RAD

- Occasionally occurs in normal individual.
- Right ventricular hypertrophy.
- Anterolateral myocardial infarction.
- Wolf-Parkinson-White (WPW) syndrome (type A).
- Dextrocardia.
- Left posterior hemiblock (Rare).

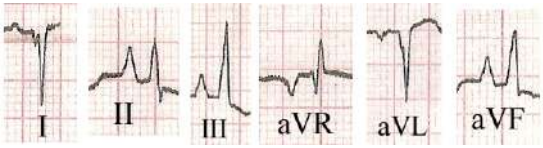


Fig.1-15. Right axis deviation. Notice the negative QRS complex in lead I and positive in lead aVF.

C| Left axis deviation (LAD)

The Left axis deviation in adults is from - 30° to - 90° (Figs. 1-14, 1-16).

Causes of LAD

- Left ventricular hypertrophy.
- Left anterior hemiblock (LAH).
- Inferior myocardial infarction.
- Wolff-Parkinson-White (WPW) syndrome (type B).

Fig. 1-16. Left axis deviation, notice the positive QRS complex in lead I and negative in lead aVF.

D| Extreme axis deviation(north-west)

The QRS is between -90° and -180° (Fig. 1-14. and Fig. 1-17), which may occur in ventricular tachycardia.

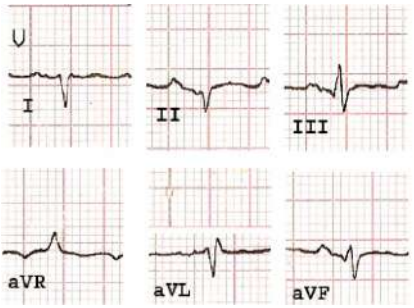


Fig. 1-17. Extreme axis deviation. Notice the negative QRS complex in lead I and in lead aVF.

E| Indeterminate axis

In the absence of the dominant QRS deflections, as in biphasic QRS complex in all limb leads (Fig. 1-18), which may occur as, normal variant, emphysema, etc.

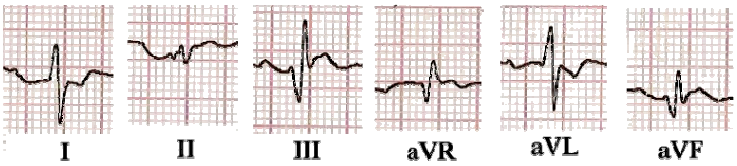


Fig. 1-18. Indeterminate axis deviation. Notice the biphasic complexes in all limb leads.

2) Transverse plane axis of the QRS complex

Commonly in normal heart position, the transitional zone (equal R and S) in the precordial chest leads starts in lead V_3 with normal progression of the R wave, i.e., a relative increase in R wave and decrease S wave amplitude from V_1 to V_6 (Fig. 1-19).

Fig. 1-19. Notice the normal transitional zone usually seen in V_3 (R = S).

The recognition of the normal R wave progression is important in the clinical practice due to the following reasons:

- Poor R wave progression (R wave does not show normal increase in amplitude through the precordial leads), e.g., anterolateral myocardial infarction.
- Reversed R wave progression is used to describe the tall R waves in right precordial leads (early transitional zone), e.g., dextrocardia and right ventricular hypertrophy.
- Absent R wave in the anterior and mid-precordial leads, with tall R wave in the lateral precordial leads, e.g., left ventricular hypertrophy.
- Sudden absence of R wave between two tall R waves in precordial leads, e.g., reversed chest leads.

Notes:

- *P and T waves axis, these measurements are rarely used in clinical practice, except where a more detailed analysis of the ECG is required.*
- *The terms clockwise and anticlockwise rotation of the heart are rarely used nowadays in clinical practice, as indicators of shifting in the transitional zone.*

Determination of the QRS Electrical Axis

The mean electrical axis of the heart is determined in the frontal plane (limb leads) by calculation of the maximal QRS deflection below and above the isoelectric line in two or more leads by using one of the following methods:

- Practical method.
- Triaxial method.
- Hexaxial method.

1) The practical method for estimating electrical axis

This method allows the reader to estimate quickly the electrical axis by looking at lead I and aVF (Fig. 1-20) as follows:

- If both of them are positive, the axis is normal.
- If lead I is negative, and lead aVF is positive this indicates right axis deviation.
- If lead I is positive and lead aVF is negative this indicates left axis deviation.
- If both are negative, this indicates extreme axis deviation.
- If both leads are biphasic (equal R and S) the axis is indeterminate.

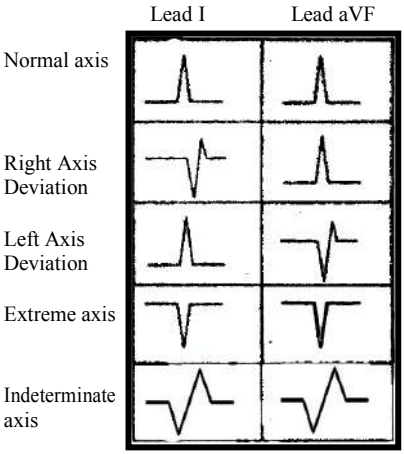


Fig. 1-20. Drawing clarifies practical method for axis determination.

Practical guidelines:

Variants of the normal heart position are:

- *Electrically horizontal heart, (commonly seen in pregnant and obese individual), in which the QRS complex is positive in lead I and biphasic in aVF.*
- *Electrically vertical heart, (commonly seen in tall individual), in which the QRS complex is biphasic in lead I and positive in aVF.*
- *You should remember that electrically vertical or horizontal heart is normal variant and not abnormal axis deviation either right or left respectively.*

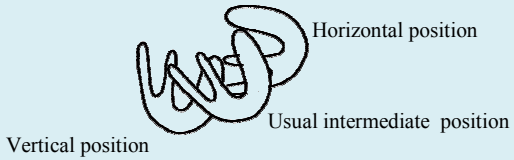


Fig. 1-21. Drawing represents the normal heart position and its variant.

2) Triaxial method of axis determination

A| Draw the triaxial reference system of the bipolar standard limb leads (I, II, III) (Fig. 1-22).

B| Determine the sum of small squares below the baseline (negative deflection of QRS) and small squares above the baseline (positive deflection of the QRS) in lead I and lead III (e.g.,1-1).

C| The net magnitude of QRS is put on the two selected leads in step B.

D| Draw perpendicular lines at the selected points, on the selected leads.

E| Draw a line from the central point of the triaxial reference system through intersection of the perpendicular lines, which is the approximate QRS axis in the frontal plane.

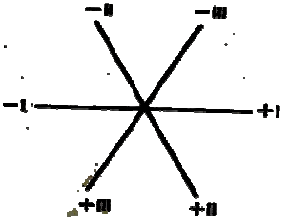
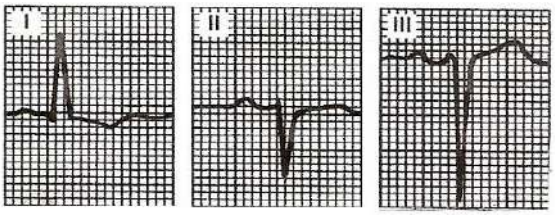


Fig. 1-22. Triaxial reference system of standard limb leads (I, II, III).

(Example 1-1)

Based on the triaxial reference system, what is the approximate mean QRS axis in this ECG?



The answer

1) The triaxial reference is drawn, which represents lead I, II, and III (Fig. 1-22).
2) lead I and lead III are used to calculate the mean QRS axis as follows:
   - A| In lead I the number of small squares below and above the baseline should be calculated (-1mm + 12mm = + 11 mm).
   - B| In lead III do the same as lead I (+1-19= -18).
   - C| Locate the net magnitude of QRS in the selected leads along the axis of these leads in the triaxial reference system (Fig. 1-23).
   - D| Draw a line from the central point of the triaxial reference system through intersection of the perpendicular lines, and its angle represents the axis of the QRS complex that located at (-45°).

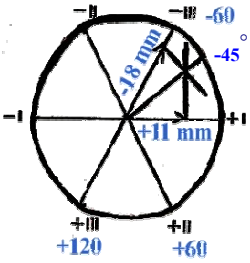


Fig. 1-23. Triaxial diagram of standard limb leads (I, II, and III).

3) Hexaxial method for estimating the electrical axis

- In this method, use the standard limb leads (I, II, III, aVR, aVL, and aVF).
- Using this method provides quick approximation of the axis, by selection of the biphasic[1] lead, then the axis is determined from the lead which is perpendicular to it, as follows:

A| Inspect all the six limb leads (I, II, III, aVR, aVL, and aVF) (example 1-2).

B| Select the lead with biphasic QRS complex (i.e. when the positive and negative deflections are equal) or the lead with smallest QRS complex.

C| Select the lead, which is perpendicular to the previously selected lead.

D| Consider the angle of this perpendicular lead it is the angle of the electrical axis (±20 degree) with reference to the hexaxial reference system (Fig. 1-24).

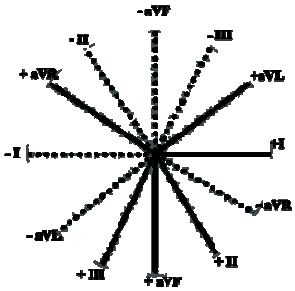
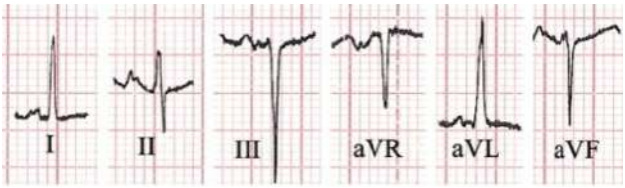


Fig. 1-24. Hexaxial reference system of standard limb leads (I, II, III, aVR, aVL, aVF)

(Example 1-2)

Based on the hexaxial reference system leads (I, II, III, aVR, aVL, aVF), what is the approximate mean QRS axis in the following ECG?



Biphasic lead: is the lead with equal positive and negative deflections, which means the QRS complex of this lead is zero net amplitude.

1) After careful inspection of the above six limb leads, we found lead II with biphasic QRS complex (i.e., the number of small squares above baseline is equal to the number of small squares below the baseline).
2) aVL is the lead that is perpendicular to lead II (Fig. 1-24).
3) Inspection of lead aVL of the above ECG, the QRS complex is dominantly upright, the QRS axis must be directed toward the positive pole of this lead which is located at (–30°) with reference to the hexaxial system (Fig. 1-14).
4) Thus the mean frontal QRS axis in this case is directed at -30° (Fig. 1-25).

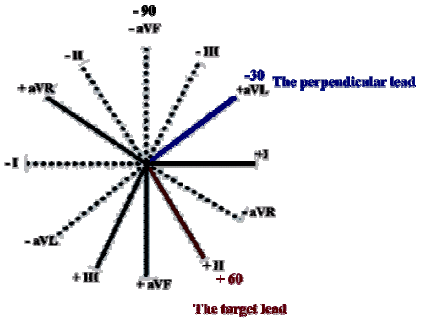


Fig. 1-25. Lead II is the target lead, and Lead aVL is the perpendicular lead

Notes:

- *If the biphasic complex is not present in limb leads of ECG strip, select the lead with smallest QRS complex.*
- *While using the hexaxial method, remember that the positive pole of aVL and aVR lie in the negative filed of the circle* (Fig. 1-14).

CHAPTER 2

Systematic interpretation of an electrocardiography

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Systematic ECG interpretation

The following points are important to consider during ECG interpretation:

A | Do not take a decision on a single ECG result particularly if the result does not fit with the clinical findings.

B | ECG abnormalities may be seen in normal healthy person, in the absence of an organic heart disease. These ECG findings include the followings:

1) Early repolarization (See Fig.7-7).
2) High left ventricular voltage (Fig. 3-2).
3) Juvenile T wave (Fig. 2-7).
4) Athletes T wave, peaked or inverted T wave seen in the precordial and limb leads.
5) Insignificant Q waves in leads I, aVL, V_5 and V_6 (See chapter 6).
6) Right axis deviation (See chapter 1).
7) Low voltage QRS complex in obese individual (See chapter 7).
8) Short PR interval (discussed later in this chapter).
9) Sinus bradycardia, first degree and Wenckbach AV block (particularly, athletes when the vagal tone is increased at rest).
10) Sinus arrhythmia (see fig. 4-2).
11) SI, SII, SIII syndrome (S wave in leads I, II, III)
12) Incomplete right bundle branch block may be seen in young patients.

C | ECG may be normal or uninterpretable in the presence of an organic heart disease. The following conditions should be considered:

1) Acute myocardial infarction may not show diagnostic ST-T changes especially in early presentation, or the myocardial infarction may be masked to be appropriately diagnosed, because of the left bundle branch block, WPW syndrome with preexcitation, the left ventricle hypertrophy, or the electronic ventricular pacemaker.
2) Patients with severe coronary artery disease may not show diagnostic ECG changes during stress test.
3) With acute pulmonary embolism, the ECG may be normal or with non-specific changes.
4) In some cases with significant left and/or right ventricular hypertrophy, the ECG may look normal.
5) The ECG may be normal between the attacks of intermittent arrhythmias such as:
   - Paroxysmal atrial fibrillation.
   - Paroxysmal supraventricular tachycardia.
   - Ventricular tachycardia and bradycardia.

D | ECG abnormalities may be seen secondary to extracardiac disease; however, a healthy heart that provides important clues in the detection of such medical conditions will be described in detail in chapter on miscellaneous ECG changes. Some of these diseases are as follows:

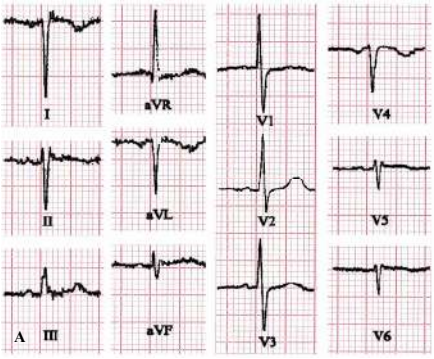
1) Cerebrovascular accident (especially intracranial hemorrhage).
2) Drug toxicity.

3) Electrolyte disorders.
4) Endocrine disorders.
5) Hypothermia.

E | Always bear in mind the following mistakes when interpreting ECG:

1) Reversed limb leads

Reversal of the left and right arm electrodes can cause right axis deviation (RAD). As a general rule, when lead I and aVL show a negative P-QRS-T waves, reversal of the left and right arm electrodes should be suspected. This is called technical dextrocardia, to differentiate it from true dextrocardia, chest leads should be studied. In reversal limb leads (technical dextrocardia) chest leads will be normal, while in true dextrocardia, chest leads will show reversed R wave progression (prominent R wave in V_1 progressively decreases in amplitude through the precordial leads) (Fig. 2-1 A and B).



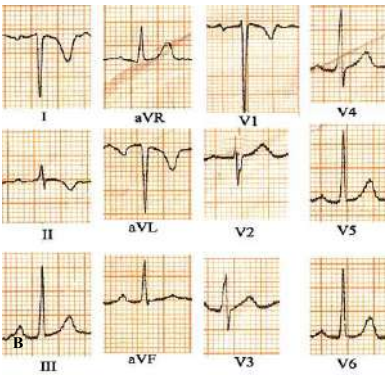


Fig. 2-1. A- Dextrocardia with situs inversus, the ECG shows a negative P, QRS and T in lead I and aVL reversed R wave progression across chest leads.

B- Limb lead reversal (technical dextrocardia), the ECG shows a negative P, QRS and T in lead I and aVL but with normal R wave progression across chest leads.

2) Reversed precordial leads

Reversal of precordial leads is rare but can cause incorrect interpretation of the ECG (Fig. 2-2).

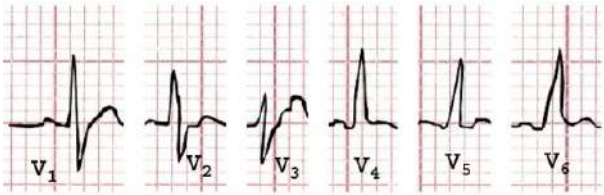


Fig. 2-2. Reversal of precordial leads V_1 and V_3, notice the abrupt decrease of R wave amplitude in V_3.

3) Unusual standardization

The normal standardization is 1 mV makes the recording needle moves vertically 10 mm = 2 large squares. Many ECGs are mistakenly thought to be "low" or "high" voltage while the voltage is actually normal, but the standardization marker is set at half or double normal standardization. Every ECG includes a calibration mark so that the gain setting can be checked. Sometimes, it is necessary to alter the calibration (Standardization), particularly when the QRS complexes appear too high or too low. It is good practice to record this clearly by writing a note on the ECG (See chapter 1, Fig. 1-8).

4) Unusual paper speed

The standard ECG recording speed is 25 mm/sec, so that 1 small square (1mm) equal to 0.04 sec, if the paper runs at double speed (50 mm/sec), the waves will double in width. If non-standard (double or half) paper speed is used. It is good practice to document this clearly at the top of the ECG (See chap. 1, Fig. 1-7).

5) External electrical interference

Wide, dark baseline artifact on the ECG is produced most often by electrical equipment that contains electric motors, such as intravenous pumps, ventilators, beds, mobile, and so on. You can eliminate these interferences by switching the ECG plug to a different outlet or turning off the other electrical device in the room (Fig. 2-3).

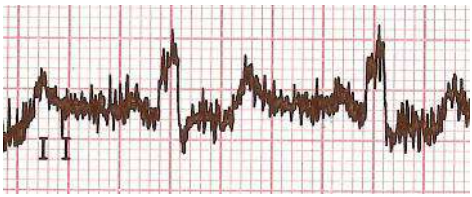


Fig. 2-3. Common ECG artifact produced by 60 Hz electrical interference.

6) Patient's movement

Skeletal muscle activity is also picked-up on the ECG (Fig. 2-4), and it is important for the patient to lie still and relaxed, while ECG is recorded. Unfortunately, this is not always possible, particularly if the patient is:

- Uncooperative or irritable.
- With respiratory distress.
- Suffering from a movement disorder.

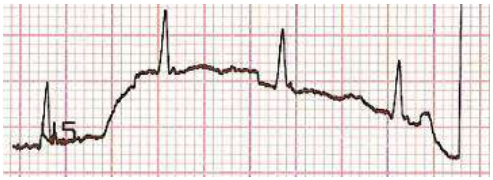


Fig. 2-4. Wandering baseline resulting from patient's movement.

7) Improper electrode fixation

Poor electrode contact, loose electrodes, broken cables, or broken wire cause this pattern (Fig. 2-5), which has a wandering baseline with unusual wavelike fluctuations. This type of artifact is simple to eliminate by either; replacing the electrodes, or the broken wires, or both.

Fig. 2-5. Unusual wavelike fluctuations

8) Unusual patient's position

The usual position of ECG recording is the supine position. Change in position on either side or elevation of the chest may affect the accuracy of recording an ECG, due to the change in the position of the heart within the chest (imagine, in the sitting position, the heart is hanging more vertically down changed in relation to the usual position of chest leads resulting in relatively low R wave amplitude). It is good practice to write the altered position on the ECG paper.

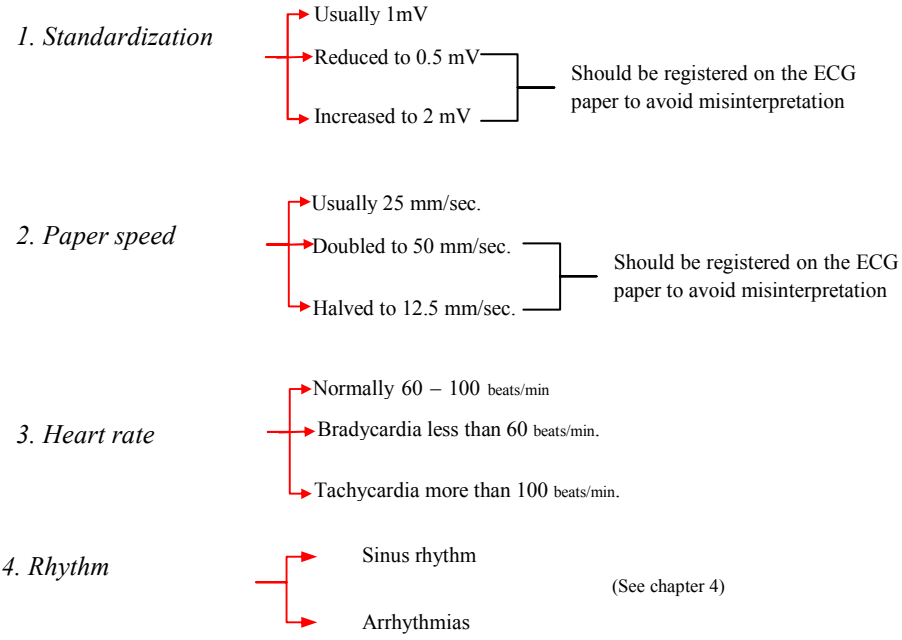
9) Artifact due to tremor

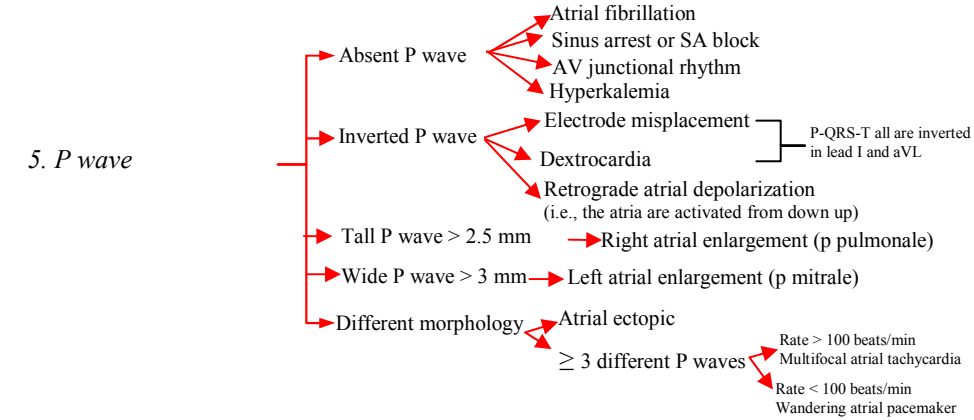
It results from pathological or physiological tremor, causing artifact on ECG as fine oscillation of baseline similar to atrial fibrillation, by looking at leads II and V_1 help to identify sinus rhythm.

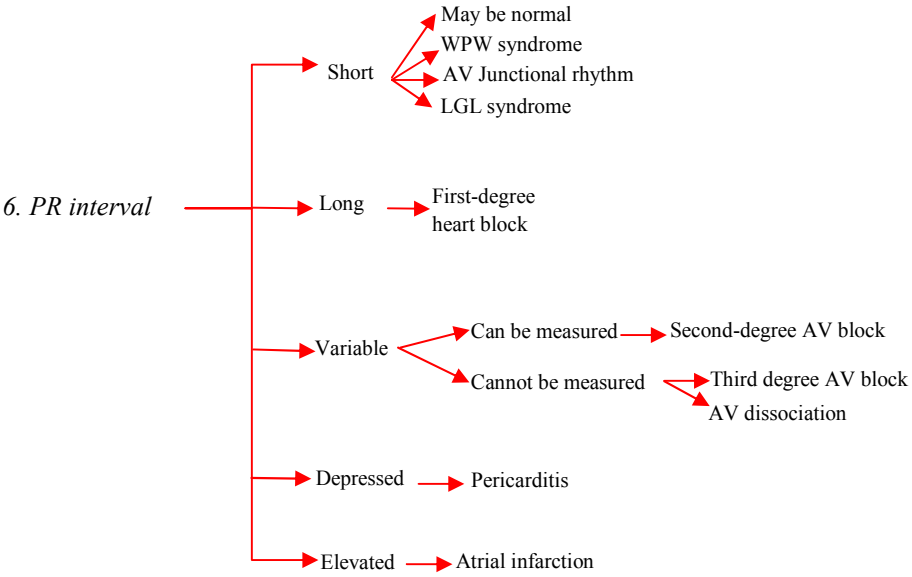
How to read an ECG

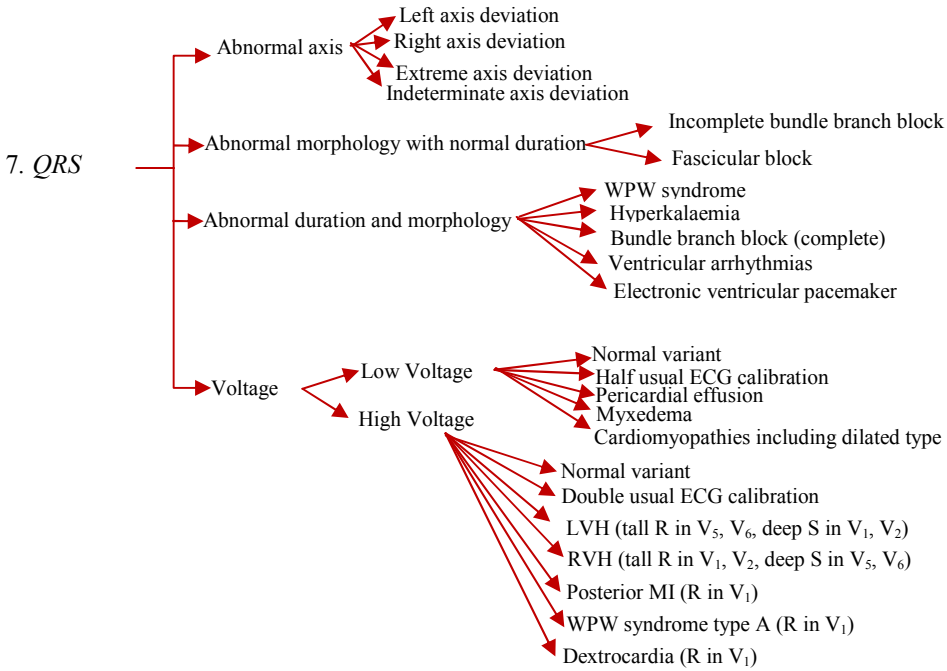
ECG interpretation is largely a matter of experience and systematic analysis. This is most easily performed by answering number of questions in logical sequence about P, QRS and T waves, segments and intervals in turn.

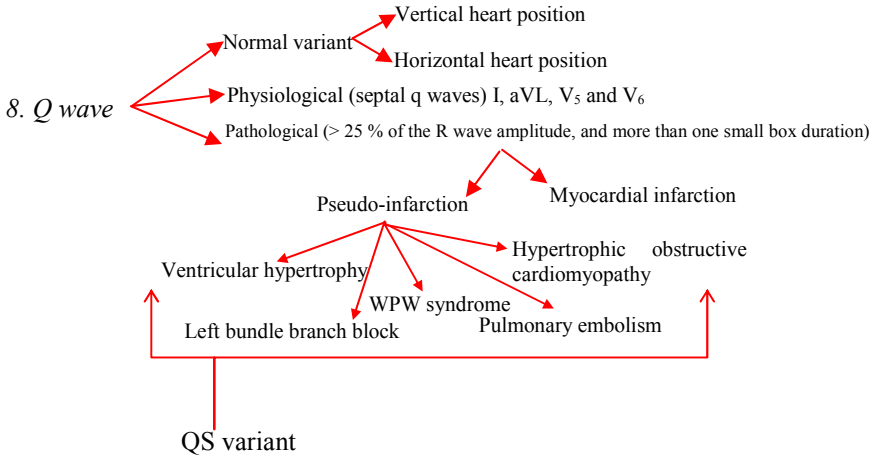
A simple system is presented in the following sequence

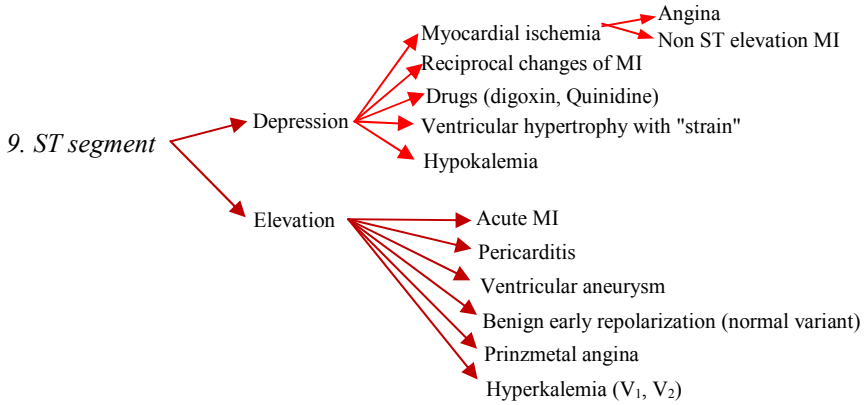


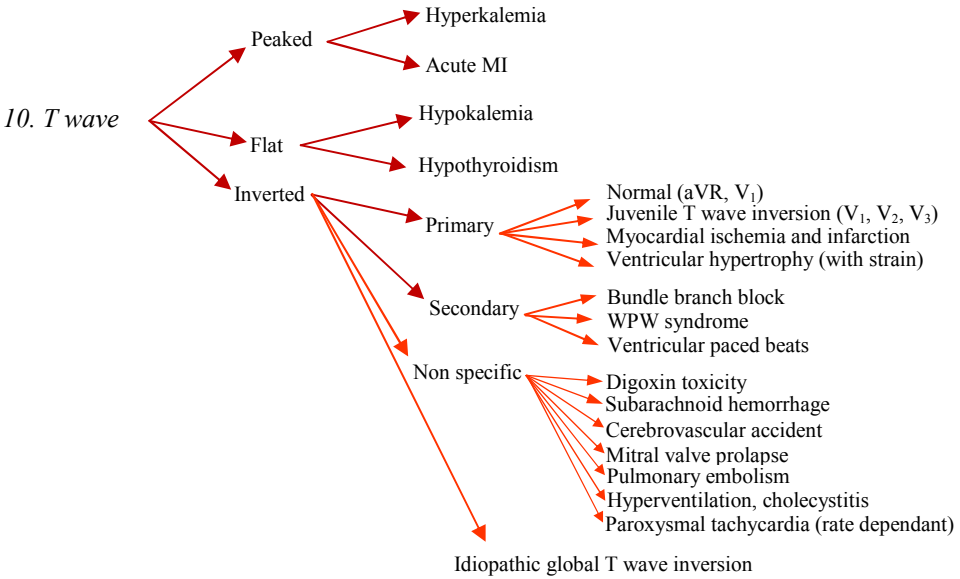

5. P wave
Absent P wave
Atrial fibrillation
Sinus arrest or SA block
AV junctional rhythm
Hyperkalemia
Inverted P wave
Electrode misplacement
Dextrocardia
P-QRS-T all are inverted in lead I and aVL
Retrograde atrial depolarization
(i.e., the atria are activated from down up)
Tall P wave > 2.5 mm
Right atrial enlargement (p pulmonale)
Wide P wave > 3 mm
Left atrial enlargement (p mitrale)
Different morphology
Atrial ectopic
≥ 3 different P waves
Rate > 100 beats/min
Multifocal atrial tachycardia
Rate < 100 beats/min
Wandering atrial pacemaker

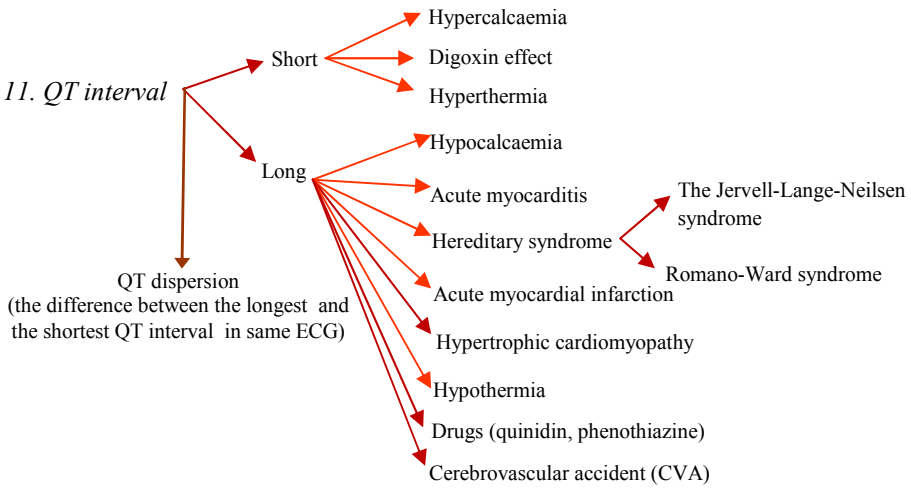

6. PR interval
Short
May be normal
WPW syndrome
AV Junctional rhythm
LGL syndrome
Long
First-degree heart block
Variable
Can be measured
Second-degree AV block
Cannot be measured
Third degree AV block
AV dissociation
Depressed
Pericarditis
Elevated
Atrial infarction

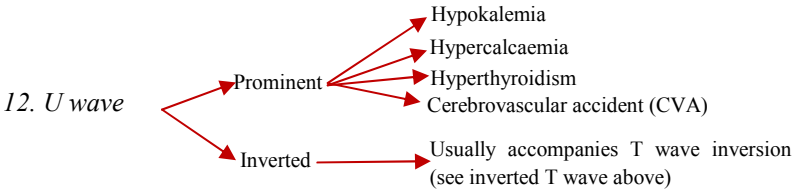

7. QRS
Abnormal axis
Left axis deviation
Right axis deviation
Extreme axis deviation
Indeterminate axis deviation
Abnormal morphology with normal duration
Incomplete bundle branch block
Fascicular block
Abnormal duration and morphology
WPW syndrome
Hyperkalaemia
Bundle branch block (complete)
Ventricular arrhythmias
Electronic ventricular pacemaker
Voltage
Low Voltage
Normal variant
Half usual ECG calibration
Pericardial effusion
Myxedema
Cardiomyopathies including dilated type
High Voltage
Normal variant
Double usual ECG calibration
LVH (tall R in V5, V6, deep S in V1, V2)
RVH (tall R in V1, V2, deep S in V5, V6)
Posterior MI (R in V1)
WPW syndrome type A (R in V1)
Dextrocardia (R in V1)


8. Q wave
Normal variant
Vertical heart position
Horizontal heart position
Physiological (septal q waves) I, aVL, V5 and V6
Pathological (> 25 % of the R wave amplitude, and more than one small box duration)
Pseudo-infarction
Myocardial infarction
Ventricular hypertrophy
Hypertrophic obstructive cardiomyopathy
WPW syndrome
Left bundle branch block
Pulmonary embolism
QS variant


9. ST segment
Depression
Myocardial ischemia
Angina
Non ST elevation MI
Reciprocal changes of MI
Drugs (digoxin, Quinidine)
Ventricular hypertrophy with "strain"
Hypokalemia
Elevation
Acute MI
Pericarditis
Ventricular aneurysm
Benign early repolarization (normal variant)
Prinzmetal angina
Hyperkalemia (V1, V2)


10. T wave
Peaked
Hyperkalemia
Acute MI
Flat
Hypokalemia
Hypothyroidism
Inverted
Primary
Normal (aVR, V1)
Juvenile T wave inversion (V1, V2, V3)
Myocardial ischemia and infarction
Ventricular hypertrophy (with strain)
Secondary
Bundle branch block
WPW syndrome
Ventricular paced beats
Non specific
Digoxin toxicity
Subarachnoid hemorrhage
Cerebrovascular accident
Mitral valve prolapse
Pulmonary embolism
Hyperventilation, cholecystitis
Paroxysmal tachycardia (rate dependant)
Idiopathic global T wave inversion


11. QT interval
Short
Hypercalcaemia
Digoxin effect
Hyperthermia
Long
Hypocalcaemia
Acute myocarditis
Hereditary syndrome
The Jervell-Lange-Neilsen syndrome
Romano-Ward syndrome
Acute myocardial infarction
Hypertrophic cardiomyopathy
Hypothermia
Drugs (quinidin, phenothiazine)
Cerebrovascular accident (CVA)
QT dispersion
(the difference between the longest and the shortest QT interval in same ECG)


12. U wave
Prominent
Hypokalemia
Hypercalcaemia
Hyperthyroidism
Cerebrovascular accident (CVA)
Inverted
Usually accompanies T wave inversion
(see inverted T wave above)

Pediatric ECG

1) Before the pediatric ECG can be interpreted, the age of the child and full clinical history must be known.
2) Interpretation of the pediatric ECG is the most challenging task because of the rapid ECG changes associated with rapid growth.

The detail of pediatric ECG falls outside the scope of this book, but a few practical points are briefly mentioned here:

- Heart rate, at birth is 140 beats/min, at 1 years 120 beats/min, and by the age of 5 years 100 beats/min. Heart rate above the age limit may be a manifestation of tachyarrhythmias, e.g., WPW syndrome (Fig. 2-6). Bradycardia is unusual in a healthy child and should arouse suspicion of significant disease such as hypothyroidism, heart block.
- QRS axis, it is deviated to the right in neonates, which may shift to normal by the age of 1-5 years in most children (average 3 years) (Fig. 2-7) but may persist to adulthood (above 16 years usually the adult axis is between "-30º and + 90º"). In children, normally the QRS axis is between (0º and +140º), deviation below or above is considered abnormal left or right axis.
- P wave, upright in lead II, inverted in aVR in sinus rhythm.

  If inverted in lead II, retrograde atrial activation (not from the SA node).

  Wide >2.5 mm in left atrial enlargement.

  High >2.5 mm in right atrial enlargement.

- Q wave, normally seen in the inferior and lateral leads but <0.04 in width and <25% of the R wave amplitude in the same complex.
- PR interval, short <0.12 sec in children and up to 0.2 sec in young adults, prolonged PR interval in children signify AV block.
- R wave, tall in V_1 in most children up to the age of 5 years but may persist up to young adults.
- QRS duration, less than or equal to 0.08 sec in children up to 8 years, prolonged QRS duration in children may indicate intraventricular conduction defect.
- T wave usually inverted in leads V_1, V_2, and V_3 until the age of 7 years at least, and may persist to adolescence (juvenile T wave inversion).

  Upright T wave in these leads 'in pediatric' ECG indicates right ventricular hypertrophy.

- QTc <0.49 sec in infants and <0.44 sec in adolescents, it is rate dependent.

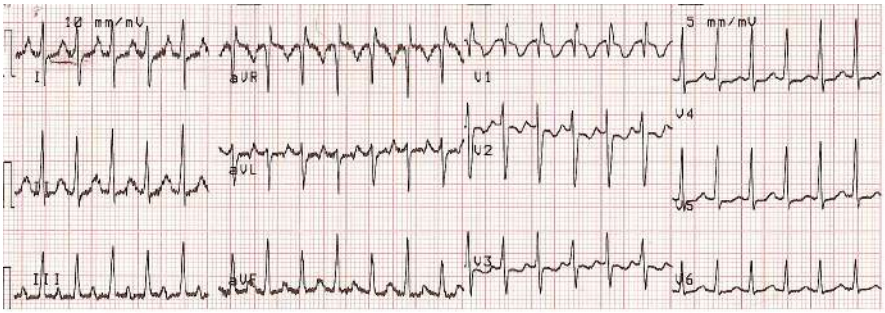


Fig. 2-6. This ECG has been taken from a child 10 year old with supraventricular tachycardia at heart rate (250 beats/min), the most common cause of SVT in this age group is an accessory atrioventricular pathway (congenital).

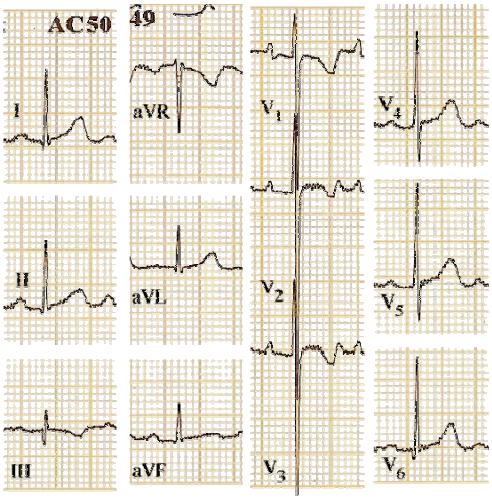


Fig. 2-7. This normal pediatric ECG obtained from 3 year old boy reveals:

- Normal axis.
- Relatively large R wave amplitude in V_1.
- Narrow QRS complex (0.08 sec.).
- Inverted T waves in $V_1 – V_3$ (Juvenile T wave)

All these findings are normal for this age group.

Note:

In pediatrics of less than one-year old, the supraventricular tachycardia is defined as heart rate of more than 220 beats/min, where as in older children more than 180 beats/min.

Source: Perry JC. Supraventricular tachycardia. In: GarsonAJr, Bricker JT, Fisher DJ, et al, editors.

The science and practice of pediatric cardiology. 2nd edition. Baltimore:Williams&Wilkins; 1998s.

CHAPTER 3

Chamber Hypertrophy and Enlargement

Left ventricular hypertrophy 54

Right Ventricular Hypertrophy 59

Biventricular Hypertrophy 62

Ventricular dilatation *(Dilated cardiomyopathy)* 63

Right atrial abnormality 64

Left atrial abnormality 65

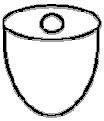
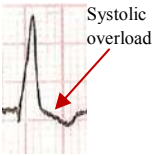
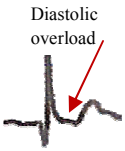
Biatrial abnormalities 66

Characteristic Combination of ECG Findings in Selected Congenital Heart Diseases 67

Chamber hypertrophy and enlargement

The ECG diagnosis of chambers hypertrophy has a low sensitivity but high specificity. Thus, approximately half of the individuals with ventricular hypertrophy cannot be recognized by the ECG (Table. 3-1).

Table. 3-1.

Normal Heart	Ventricular Hypertrophy	Ventricular Dilatation
		
Normal Heart	Concentric hypertrophy	Eccentric hypertrophy
	Characterized by increase QRS voltage, ST- depression, and T wave inversion in the left precordial leads. 	Characterized by tall late R wave in V_5 and V_6 with ST-elevation (diastolic overload) 

Left ventricular hypertrophy (LVH)

Pathophysiology of LVH

- Increased muscle thickness of the left ventricle causes shift of QRS axis toward the left precordial leads (more muscle, more voltage) that results in a deep S wave in the right oriented leads and tall R waves in the left oriented leads (Voltage criteria) (Fig. 3-1A&E).

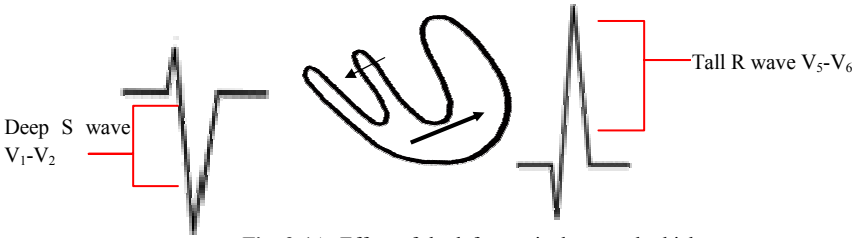


Fig. 3-1A. Effect of the left ventricular muscle thickness on the amplitude and direction of QRS complex

- When the hypertrophied left ventricle is under strain, this probably interferes with blood supply of the subendocardium causing secondary ST-T changes in the opposite main QRS direction (referred to as strain pattern) in the left oriented leads V_5 and V_6 (Fig. 3-1B and E).

Fig. 3-1B. Strain pattern (secondary ST-T changes opposite to the direction of the QRS) (arrow)

- The delay in depolarization of the hypertrophied left ventricle causes widening of QRS complex and prolongs the ventricular activation time (Fig. 3-1C).

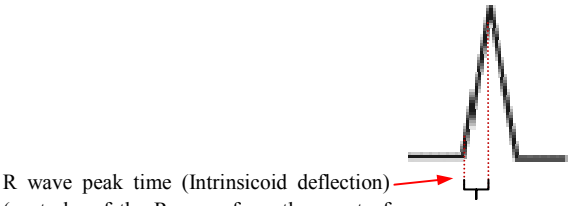


Fig. 3-1C. Ventricular activation time (from onset of QRS complex to the peak of R wave) is more than 0.05 sec. (Normally it is less than 0.05 sec).

- High left ventricular pressure is reflected on the left atrium causing atrial (P wave) abnormalities in the ECG (Fig. 3-1D, see also Fig. 3-8 B and C).

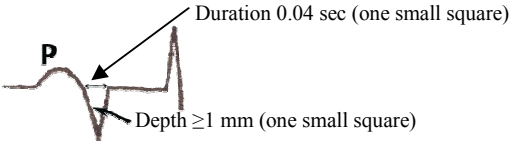


Fig. 3-1 D. Left atrial enlargement, wide biphasic P wave with negative deflection in lead V_1 > 1mm.

- Long standing hypertrophy causes fibrosis in the left anterior fascicle of the left bundle (anterior hemiblock) results in left axis deviation (LAD) (See chapter 1).
- In advanced cases, LVH causes pressure on the septum results in partial left bundle branch block. In some individuals, complete left bundle branch block appears and completely masks the ECG finding of LVH (See chapter 5).

ECG manifestation of LV hypertrophy

A | LVH both by voltage criteria and by ST-T changes

The Romhitt and Estes point score system:

- Voltage criteria (3 points)
  - R or S wave in any limb lead ≥ 20 mm.
  - S wave in V_1 or V_2 ≥ 30 mm.
  - R wave in V_5 or V_6 ≥ 30 mm.

- ST-T abnormalities (3 points) without digitalis, but if digitalis (1 point)
- P wave of left atrial enlargement (3 points)
- Left axis deviation (2 points)
- Increase R wave peak time (1 point)
- QRS duration ≥ 0.09 sec (1 point)

LVH ≥ 5 points.
Probable LVH = 4 points.

B | LVH by voltage criteria alone

Other ECG voltage criteria for diagnosis of LVH as follows:

- Cornell voltage criteria[1]:
  S in V_3 + R in aVL ≥ 24 mm (men)
  S in V_3 + R in aVL ≥ 20 mm (women)
- Sokolow-Lyon limb leads voltage criteria:
  R in aVL ≥ 11 mm.
  R in aVF ≥ 20 mm.
- Sokolow-Lyon chest leads voltage criteria:
  S wave in V_1 plus R wave in V_5 or $V_6 \geq 35$ mm.

[1] Cornell voltage criteria are the most useful in clinical practice (because both limb and chest voltage criteria are used) if other voltage criteria are invalid in the presence of disease such as anterior myocardial infarction.

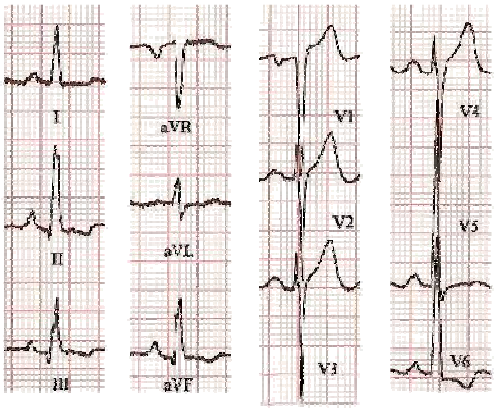


Fig. 3-1 E. Left ventricular hypertrophy, S wave in V_1 and R wave in V_6 > 35 mm, notice secondary ST-T changes, (left ventricular strain pattern).

Practical guideline:

The voltage criteria alone are not enough for the diagnosis of LVH, and this limitation is referred to the following factors:

- *High voltage QRS complex may be normal variant in young adults (Fig. 3-2).*
- *Normal ECG does not exclude LVH.*
- *In the presence of other diseases such as pericardial effusion, emphysema, and myocardial infarction, these diseases may change the voltage criteria of LVH.*

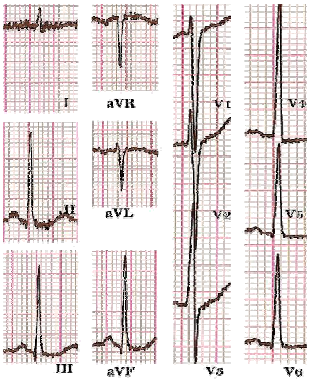


Fig. 3-2. High voltages in the chest leads (S_{V1} + R_{V5} = 36 mm) from a 20 year old man represent a common normal ECG variant. No secondary ST-T changes, left axis deviation or left atrial enlargement.

Brief review of ECG changes in hypertrophic cardiomyopathy (HCM)

LVH without identifiable cause is called hypertrophic cardiomyopathy (HCM) in which the hypertrophy is asymmetrical which may be:

- Septal HCM (deep Q waves in the inferior and lateral leads) (Fig. 3-3A).
- Apical HCM (deep inverted T waves in leads V_1 through V_6 with LVH criteria) (Fig. 3-3B).

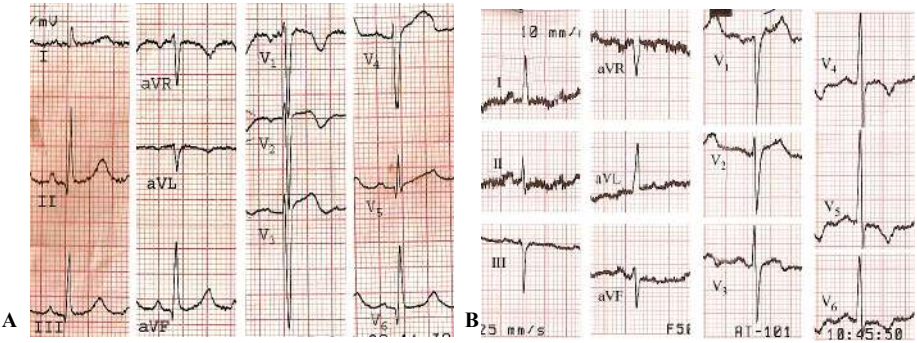


Fig. 3-3.

A. Septal hypertrophic cardiomyopathy notice the presence of Q wave in the inferior and lateral leads.

B. Apical hypertrophic cardiomyopathy, the ECG shows signs of LV hypertrophy and symmetrical T wave inversion in leads V_4 through V_6, that should be differentiated from the T wave inversion of ischemia by clinical correlation and biomarkers.

Note:
Q or QS pattern in patient with LVH simulating MI is named pseudoinfarction pattern (see chap. 2) differential diagnosis of Q wave.

Right Ventricular Hypertrophy (RVH)

Pathophysiology of RVH

- Hypertrophy of the right ventricle causes shift of the electrical forces toward the right side of the heart, resulting in high amplitude R waves in leads V_1, V_2 and deep S waves in the left precordial leads V_5 and V_6 (Fig. 3-4 A and B).
- Increased muscle thickness of the right ventricle interferes with the blood supply, causing strain pattern (ST-T changes) directed opposite to the QRS direction in the right precordial leads.

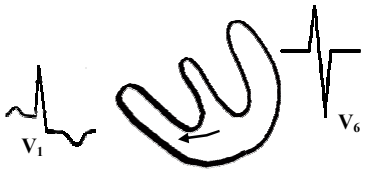


Fig.3-4A. Effect of the right ventricular hypertrophy on the amplitude and direction of QRS complex.

ECG diagnosis of Right Ventricular Hypertrophy.

Suggestive Criteria:

- R/S (ratio) in $V_1 \geq 1$, or
- R in $V_1 \geq 7$ mm, or
- R in V_1 + S in V_5 or $V_6 \geq 10.5$ mm

Supportive findings:

- Right axis deviation.
- Right atrial abnormality.
- ST depression + T wave inversion in V_1 or V_2 (RV strain).
- Normal QRS duration if there is no right bundle branch block.

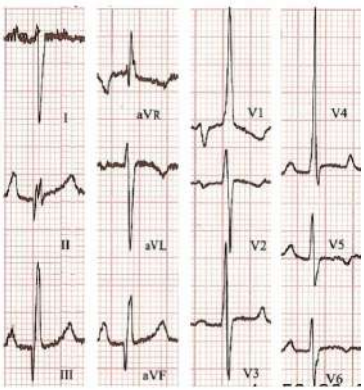


Fig. 3-4 B. Right ventricular hypertrophy with "Strain"

Notice the following ECG changes:

- R waves in lead V_1 more than 7 mm.
- R wave in the V_1 + S wave in V_6 more than 10.5 mm.
- Right axis deviation.
- ST segment depression &T wave inversion in leads V_1, V_2
- Right atrial enlargement.

Practical guidelines:

- *Tall R wave in V_1 is the most sensitive for diagnosis of RVH because of its proximity to the right ventricle, other differential diagnosis of tall R wave in V_1 are summarized in table 3-2.*
- *RVH may also be manifested as dominant S waves across all the chest leads.*
- *Deep S wave in lead I is an important clue of right ventricular hypertrophy.*
- *Other rare variant of cardiomyopathy is arrhythmogenic right ventricular dysplasia, which it characterized by replacement of right ventricular free wall by fat deposition resulting in dilatation of the right ventricle. It has the following ECG findings:*

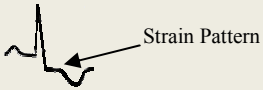
  ** In sinus rhythm*

  - Epsilon waves, T wave inversion, and increase QRS duration in V_1 through V_3.

  ** In tachyarrhythmias*

  - Ventricular tachycardia with LBBB morphology and right axis deviation because of the delayed activation from the right side.

Table 3-2. Differential diagnosis of tall R wave in V_1

N	Causes	Confirmatory clues
1	Normal variants	No other abnormalities
2	Posterior MI	ST depression, upright T wave in V_1-V_3 (See also Fig. 6-10D)
3	Right ventricular hypertrophy	Secondary ST-T changes(strain pattern), right axis deviation, right atrial enlargement (See also Fig. 3-4B) 
4	Ventricular septal hypertrophy	Associated Q wave in the inferior and lateral leads, signs of left ventricular hypertrophy (Fig. 3-3A).
5	Right bundle branch block	Wide QRS complex if complete, broad (slurred) S in V_6 (not present in RVH), delayed R peak in V_1 (See also Fig. 5-2D)
6	WPW-syndrome type A	Short PR interval – delta waves (See also Fig. 4-19)
7	Dextrocardia (mirror image)	Inverted P-QRS-T wave in lead I, with progressive decrease of R wave in the chest leads (see also Fig. 2-1A)
8	Incorrect position of the precordial leads V_1 and V_3	The reversal of chest leads is rare but can cause incorrect interpretation of ECG. Notice in Figure 2-2, reversal of V_1 and V_3 resulting in tall R wave in the V_1 and small R wave in V_3.

Modified from Marriott HJL, Glancy DL: Value of leads V7-V9 in Diagnosing Posterior Wall Acute Myocardial Infarction and other causes of tall R waves in V1-V2, Am J., Cardiol, 1997, 80:508-509.

Biventricular Hypertrophy

Combined ventricular hypertrophy is suggested by the following combination of ECG changes:

- ECG criteria for LVH in conjunction with any of the following:
  Right axis deviation
  Prominent R waves in the right precordial leads
  ECG changes of right atrial abnormality (Fig. 3-5).
  S more than R in V_6
  R more than Q in aVR
- Low amplitude S wave in lead V_1 combined with a very deep S wave in lead V_2.
- Left atrial enlargement and left axis deviation as suggestive criteria for LVH combined with any criterion suggestive of RVH.
- Kutz-Wachtel phenomenon consisting of high voltage biphasic (R=S) complexes in mid precordial leads. This is seen in many congenital lesions, most commonly in VSD.
- In rare cases, the ECG may not show any finding because of partial cancellation of electrical forces, however, clinical evidence shows biventricular hypertrophy.

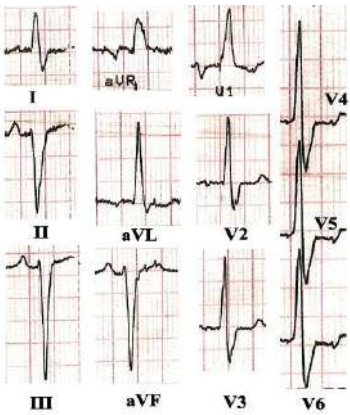


Fig. 3-5. ECG. Biventricular hypertrophy, notice the prominent R wave in V_1 and deep S wave in V_6, R more than Q in aVR, biphasic P wave in V_1, and left axis deviation.

Ventricular dilatation (Dilated cardiomyopathy)

The dilated cardiac chambers with impaired systolic function, which can be primary (idiopathic), or secondary (e.g., ischemic).

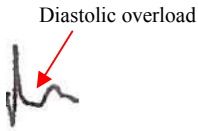
Diagnostic ECG criteria

ECG signs of LVH with one or more of the following ECG findings (Fig. 3-6):

1) Low voltage QRS complexes in limb leads.
2) Slow progression of R waves in chest leads.
3) The R wave is taller in lead V_6 than V_5.
4) Small S wave in V_1 which results in small biphasic rs complex combined with deep S wave in V_2.

Note:

The combination of the above ECG changes is commonly seen in congestive heart failure



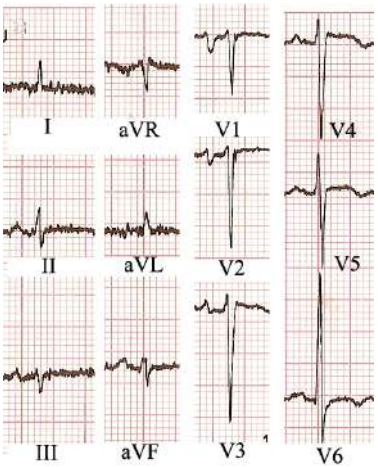


Fig. 3-6. Left ventricular hypertrophy and dilatation (eccentric hypertrophy).

Notice the following ECG changes:

- Low voltage QRS.
- Complex in the limb leads.
- Slow R wave progression in $V_1 - V_4$.
- High voltage QRS in lateral leads.

Practical guideline:

Primary (idiopathic) dilated cardiomyopathy is essentially a diagnosis of exclusion. Secondary causes (e.g. ischemic DCM) should be identified and treated.

Atrial Abnormalities

1. Right atrial abnormality

Dilatation of the right atrium shifts the electrical axis downward in the direction of the positive poles of the inferior leads II, III, and aVF (Fig. 3-7A), causing high amplitude P wave without delay in depolarization ,thus the duration of the P wave is not prolonged.

Fig. 3-7 A. Arrow indicates the direction of electrical activity in right atrial enlargement.

ECG changes of right atrial abnormality

1) Tall, peaked P wave (P Pulmonale) ≥ 2.5 mm in leads II, III and aVF (Fig. 3-7B).
2) Initial upward positive amplitude of P wave in V_1 ≥1.5 mm (Fig. 3-7 C).
3) Normal P wave duration.

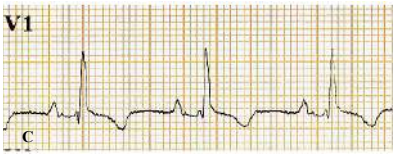


Fig. 3-7 B, Right atrial abnormality. Tall, peaked P wave in lead II. **C**, initial positive P wave in V_1 more than 1.5 mm.

2. Left atrial abnormality

The delay in depolarization of the dilated left atrium (Fig. 3-8 A) prolongs the P wave duration.

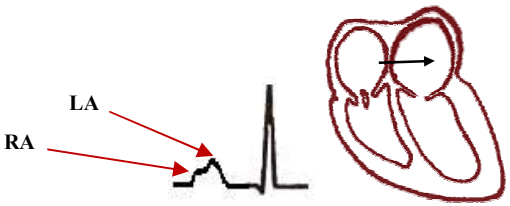


Fig. 3-8A. Arrow indicates direction of electrical activity in left atrial enlargement, the P wave is bifid, the first peak represents right atrial depolarization and the second for the left atrial enlargement.

ECG changes of left atrial abnormality

1) Increased P wave duration ≥ 0.12 sec. (3 small squares) (Fig. 3-8 B).
2) downward terminal negative deflection of P wave in V1 ≥ 1mm in depth, with duration ≥ 1mm (Fig. 3-8 C).
3) Wide, notched P wave (P mitrale) in leads II, III and aVF.

Fig. 3-8. B. Broad notched P wave in lead II.
C, Biphasic P wave in lead V_1; the negative deflection > 1 mm deep and wide.

***Comments**:*

- *Left atrial enlargement and left axis deviation are suggestive signs of LVH in the presence of LBBB.*
- *Left atrial enlargement may be the earliest and the only ECG sign of the systemic hypertension.*

3. Biatrial abnormalities

- Biatrial enlargement is suggested by the combination of the following ECG changes:
  1) Peaked, tall P wave ≥ 2.5 mm and duration ≥ 3 mm in limb leads most apparent in lead II (Fig. 3-9 A and B).
  2) Biphasic P wave in V_1 with initial positive amplitude ≥ 1.5 mm, and negative deflection ≥ 1mm in depth and duration (Fig. 3-9 C and D).

     Peaked positive component and deep negative component of P wave in V_1 are due to RA, LA abnormalities respectively

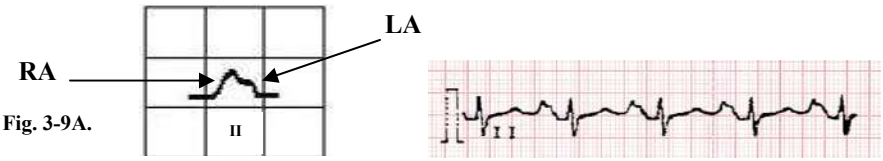


Fig. 3-9A.

Fig. 3-9 A and B. Biatrial hypertrophy, wide notched P wave

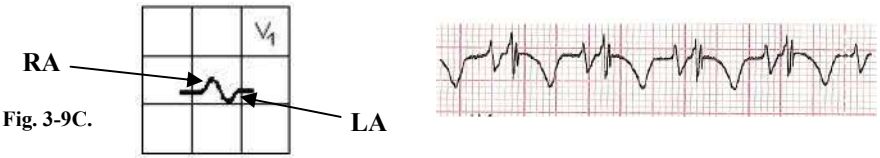


Fig. 3-9C.

Fig. 3-9C and D. Biatrial hypertrophy, peaked positive and deep negative component of P wave in V_1.

Characteristic Combination of ECG Findings in Selected Congenital Heart Diseases

There are certain congenital heart diseases associated with typical combinations of abnormalities on the ECG; some of these diseases are summarized in Table 3-3.

Table 3-3.

	Type	ECG findings
1	Dextrocardia with situs inversus.	P, QRS, and T inversion in lead I and aVL with right axis deviation
2	Anomalous left coronary artery originating from the pulmonary artery	Q wave, ST elevation, and T wave inversion in leads I, aVL, V4, V5, and V6.
3	Ostium primum atrial septal defect	the ECG shows left axis deviation and right bundle branch block (Fig. 3-10).
4	Ventricular septal defect with pulmonary artery hypertension/or stenosis,	biventricular hypertrophy characterized by high voltage biphasic QRS complexes in the midprecordial leads
5	Ebstein's anomaly	Very large P wave (Himalaya P wave). Very wide QRS complex and bizarre shape. Absence of the left ventricular activity

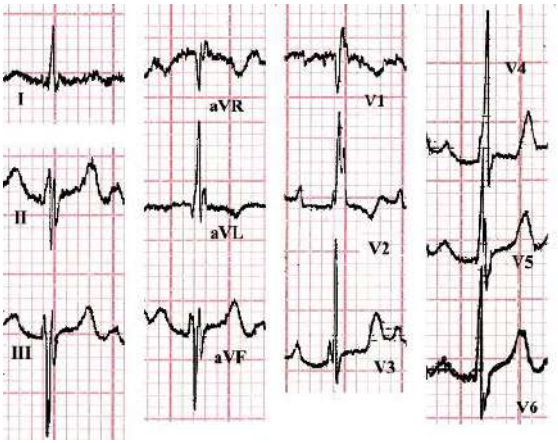


Fig. 3-10. A 60 year old female with an ostium primum atrial septal defect.

Practical guidelines:

- *Despite the ECG is not sensitive for detection of cardiac chambers hypertrophy or enlargement, the clinical significance of identification their ECG findings are important for treatment and prognosis. For example left atrial enlargement and LVH in case of systemic hypertension, aortic stenosis, and (HCM). Left ventricular dilatation in case of valvular regurgitation, and ischemic heart disease (ischemic cardiomyopathy).*
- *Left sided heart disease, pulmonary diseases may result in right atrial enlargement and right ventricular hypertrophy with consequences elevation of pulmonary artery pressure that consider as an indicator of advanced long-standing diseases.*
- *Remember the mitral stenosis may produce a combination of left atrial enlargement and RVH*

CHAPTER 4

Cardiac Rhythm Disorders

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Cardinal Features of Normal Sinus Rhythm (NSR)

To recognize the abnormal rhythm, the characteristic features of the normal sinus rhythm (Fig 4-1), should be remembered:

1) P waves are upright in lead II and inverted in lead aVR.
2) Every P wave is of similar shape and axis.
3) PR interval is fixed between 3-5 small squares.
4) Every QRS is preceded by P wave.
5) RR interval is regular, however; sometimes it has very slight irregularity.
6) Heart rate is between 60-100 beats/min.

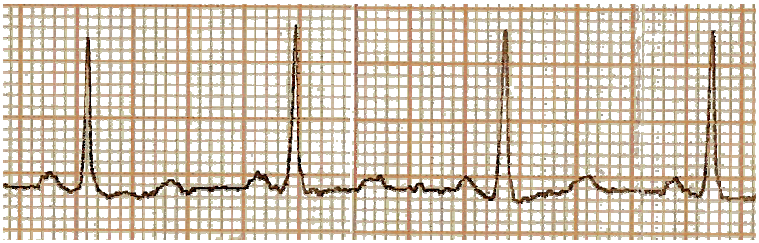


Fig. 4-1. Sinus rhythm, the P wave is upright, appears before QRS complex with normal heart rate.

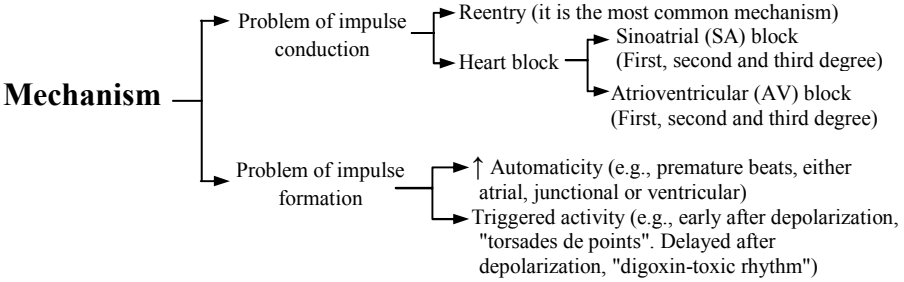
Abnormal Cardiac Rhythm (arrhythmia or disarrhythmia)

Definition

Any deviation from normal sinus rhythm is named an arrhythmia.

Causes

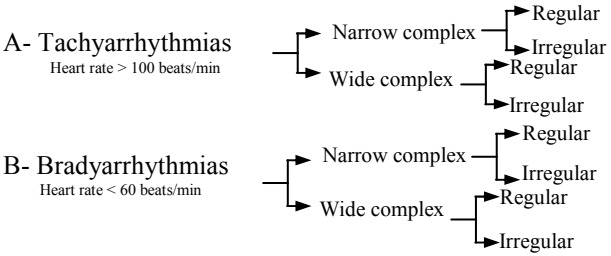
Heart disease, hypertension, pulmonary disease, hyperthyroidism, poisons, drugs, alcohol, and metabolic disturbance can cause arrhythmias, some people are congenitally prone to arrhythmias. Thus, finding the cause of arrhythmia is important because the treatment depends on the underlying cause.



Classification

The heart arrhythmias can be classified according to their effect on the heart rate, or on the site of origin.

I. According to their effect on the heart rate



Tachy and bradyarrhythmias are discussed briefly at the end of this chapter.

II- According to the site of origin

Arrhythmias originating in the sinus node (SA nodal arrhythmias)

1. Sinus arrhythmia

- Normal sinus P waves and PR intervals.
- Irregular PP and RR intervals; the variation between the shortest and longest interval is > 0.16sec (four small squares) (Fig 4-2).
- Every P wave is followed by QRS complex, i.e., 1:1 AV conduction.
- It is a normal finding in children and young adults and it tends to disappear with advanced age.
- During the long intervals of sinus arrhythmia, junctional escape beats may occur.
- In the phasic variety, the SA nodal rate accelerates during inspiration and decelerates during expiration.

Fig. 4-2. Sinus arrhythmia, the variation between the shortest and longest R-R interval is > 0.16sec.

2. Sinus tachycardia

It is a sinus rhythm with heart rate above 100 beats/min (Fig 4-3).

Fig. 4-3. Sinus tachycardia with heart rate 110 beats/min

***Note**:*
Tachycardia in a sleeping patient may be caused by
- *Fever*
- *Bleeding*
- *Thyrotoxicosis*

3. Sinus Bradycardia and its variants

A | Sinus bradycardia

It is a sinus rhythm with heart rate less than 60 beats/min (Fig 4-4A).

Fig. 4-4 A. Sinus Bradycardia with heart rate 50 beats/min.

B | Sinoatrial block

Intermittent failure of sinus impulse conduction, causes an entire P-QRS-T cycle to be dropped (nothing present on the baseline), the pause is double the P-P interval between two sinus beats (Fig. 4-4B).

SA block is divided in to three types, the first degree SA block is difficult to recognize on the surface of ECG, second degree SA block is manifested as long P-P interval, the third degree SA block can not be distinguished from the sinus arrest.

Fig. 4-4 B. Second-degree sinoatrial exit block. Surface ECG denoting abrupt absence of P-QRS-T cycle between the sinus rhythm (arrow) and the P-P interval of the blocked beat is double.

C | Sinus arrest

Intermittent failure of sinus impulse formation causes drop of P-QRS-T complexes resulting in pause more than double the P-P interval between two sinus beats (Fig. 4-4 C); and usually manifested as bradycardia followed by compensatory tachycardia in the form of atrial fibrillation or atrial flutter which is named, brady-tachy arrhythmias (sick sinus syndrome).

Fig. 4-4 C. Sinus arrest results from failure of impulse formation resulting in pause > 2 sec.

***Practical guideline**:*

In clinical practice, hard work should be done to identify P wave including preceding T wave to make differential diagnosis of ECG pause, which is most commonly caused by the blocked atrial premature complex or second degree AV block.

D | Bradycardia caused by increase vagal stimulation

- Carotid sinus syndrome

  Usually occurs in elderly people, it may cause sinus bradycardia by stimulation of the carotid sinus associated with turning of the neck, wearing stiff collars, or coughing.

- Neurocardiogenic (vasovagal) syndrome

  Bradycardia, usually affects young adults but it may occur for the first time with elderly people.

Arrhythmias originating in the atria (Atrial arrhythmias)

1. Atrial premature beat

- P wave of the ectopic beat has different shape and axis and it appears immediately (prematurely) before the next expected beat (Fig 4-5).
- The rate is usually less than 100 beats/min.
- PP intervals are usually irregular because of ectopic beats.
- PR intervals are usually normal, but may be increased or decreased.
- QRS is narrow unless there is an abnormal conduction.
- Ectopic atrial beat is usually followed by incomplete compensatory pause, in contrast to the ventricular premature beat, which is followed by complete compensatory pause.

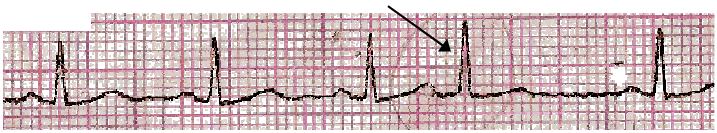


Fig.4-5. Atrial premature beat (arrow). Its QRS complex is identical to those of normal beats but the P wave slightly differs in shape and deforms T wave of the preceding beat.

2. Multifocal Atrial Tachycardia (MAT)

- Impulses are originated irregularly and rapidly at different points in the atria resulting in different P waves morphology (≥ 3 different P waves) (Fig 4-6 A and B).

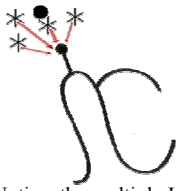


Fig. 4-6 A. Notice, the multiple Impulses are originated irregularly and rapidly at different points in the atria.

- PR, PP and RR intervals are variable.
- Atrial rate 100 -180 beats/min (average 150 beats/min).
- Almost exclusively associated with severe pulmonary disease and theophylline toxicity.

Fig. 4-6 B. Multifocal atrial tachycardia, notice different P wave morphology (arrows), heart rate > 100 beats/min.

3. Wandering atrial pacemaker (WAP)

- The impulses originate from different points in the atria resulting in different P waves morphology similar to that of multifocal atrial tachycardia, but with heart rate <100 beats/min (Fig. 4-7).
- PR, PP, and RR intervals are variable.
- Generally, it is benign rhythm.

Fig. 4-7. Wandering atrial pacemaker, notice the different P wave morphology (arrows), heart rate < 100 beats/min.

4. Atrial Fibrillation (AF)

- **Definition**

  It is unorganized atrial activity with completely irregular ventricular rate.

- **Types**

  It is the most common sustained arrhythmia, occurs as:

  A | Paroxysmal pattern (episodic AF that terminates to normal rhythm spontaneously).

  B | Persistent pattern (episodic AF that terminates only after an intervention).

  C | Permanent pattern (continuous AF that resists attempts to restor sinus rhythm).

  Another recent classification of atrial fibrillation is:

  New onset, recurrent, and permanent. (ACCSAP$_{version}$7, 2009)

- **Pathophysiology**

  There are two theories:

  One theory proposed that enhanced automaticity involving one or more foci firing rapidly (Fig 4-8A).

  The other is based on hypotheses of multiple re-entrant wavelets.

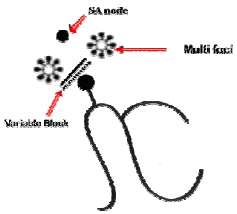


Fig. 4-8 A. Notice unorganized multiple atrial foci firing rapidly.

- **The ECG characteristics of AF are** (Fig. 4-8 B and C):

  A | Absent P waves, the isoelectric line is replaced by fibrillatory waves (f waves).

  B | Coarse or fine undulation of the ECG is baseline commonly seen in lead V_1.

  C | The QRS complexes vary in amplitude.

Fig. 4-8.
B. coarse atrial fibrillation with undulated baseline as fibrillatory (f) waves.
C. fine atrial fibrillation : the fibrillatory (f) waves are hard to identify, but the irregular R-R interval and variable R wave amplitude help to diagnose the atrial fibrillation.

D | Ventricular rhythm is quite irregularly irregular, slow (Fig 4-8D) or rapid (Fig 4-8E), because the ventricles are stimulated in random fashion due to variable block of rapid atrial impulses at the AV node (in case of atrial flutter the block is usually constant).

Fig. 4-8 D. Atrial fibrillation with irregular slow ventricular response. Notice the oscillation of the baseline due to fibrillatory (f) waves, no true P waves.

Fig. 4-8 E. Atrial fibrillation with irregular rapid ventricular response with relatively subtle wrinkly appearance f waves between irregular R waves.

- **Differential diagnosis**
  - Atrial flutter with variable block, the flutter waves (saw-toothed) are more prominent than fibrillatory (f) waves, especially in the inferior leads and V_1.
  - Multifocal atrial tachycardia (P waves have three or more different morphology).
  - Atrial tachycardia with AV block, the non-sinus P wave can be easily identified in the baseline.

Practical guidelines:

- *In the setting of atrial fibrillation, if the ventricular rate has become regular, it suggests one of the following possibilities:*
- *Conversion to sinus rhythm.*
- *Conversion to atrial flutter.*
- *Complete AV block (the ventricular rate is regular).*
- *Digitalis toxicity (Fig .4-8F).*
- *Unexplained AF (or sinus tachycardia at rest) should prompt a search for hyperthyroidism.*
- *The more atrial enlargement, the more persistent atrial fibrillation.*
- *In young patients (especially with no evidence of heart disease), the atrial fibrillation usually occurs in paroxysm called Lone atrial fibrillation.*
- *Remember, the atrial fibrillation licks the heart and bites the brain by the thrombo-embolic stroke, thus; anticoagulant are the mainstay of treatment beside the rate controlling drugs.*

Fig. 4-8 F. Atrial fibrillation and digitalis effect, notice the slow and regular ventricular rate because of the complete AV block.

Ashman syndrome

A pattern of short RR interval followed by a long RR interval occurs in the setting of SVT including atrial fibrillation with aberrant conduction. The morphology of QRS that simulates RBBB pattern, absence of compensatory pause, and relatively narrow QRS may help to differentiate it from ventricular ectopic, which is important in treatment and prognosis (Fig. 4-9).

Fig. 4-9. Notice the rhythm is irregularly irregular, the occasional wide complex aberrantly conducted supraventricular beat occurring after a QRS that is preceded by long pause the long cycle results in delayed repolarization. Named Ashman syndrome.

5. Atrial Flutter

- **Definition**

  It is a tachyarrhythmia produced by macro-reentrant wavelets within a single circuit in the atria and characterized by a distinct saw-toothed flutter waves in the inferior leads and sharp (prominent) flutter waves in lead V_1, with no clear isoelectric line, and relatively constant AV block 2:1, ventricular rate 100 – 150 beats/min.

  - It is the second most common tachyarrhythmia.
  - It is usually paroxysmal, lasting from seconds to hours and even to days in some cases.

- **Pathophysiology**

  It results from macro-reentrant wavelets in the right atrium, but rarely of left atrial re-entry.

- **Atrial flutter can be classified into:**

A | Typical atrial flutter

It is the most common type of atrial flutter. The re-entrant impulses travel counter-clockwise as regular rotation (down the right atrial free wall and up to the interatrial septum) (Fig 4-10A).

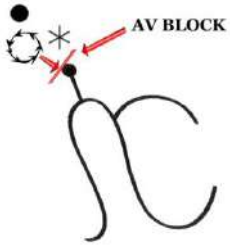


Fig. 4-10 A. Notice the firing focus with counter-clockwise rotation of the impulses in the atria, with blocking one out of two atrial impulses (2:1), two of three (3:1), or three of four (4:1).

B | Atypical atrial flutter

The re-entrant impulses travel clockwise as a regular rotation (Fig 4-10B).

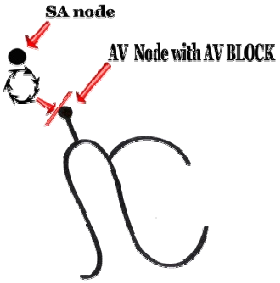


Fig. 4-10 B. Notice the firing focus with clockwise rotation of the impulses in the atria. The AV node acts as a gatekeeper to prevent those rapid impulses from reaching the ventricles by blocking one out of two atrial impulses (2:1), two of three (3:1), or three of four (4:1).

- **The ECG characteristics** (Fig 4-10C)
  - Typical flutter shows the classic negative saw-toothed P waves in the inferior leads, and positive in V_1.
  - Atypical flutter shows positive saw-toothed P waves in the inferior leads, and negative in V_1.
  - QRS complexes have normal duration, although aberrant conduction could occur.
  - The ventricular response is typically regular, but it can be irregularly irregular due to varying degrees of AV block (2:1, 3:1, etc).
  - If the flutter (F) waves are not clearly visible, they can be clarified by slowing flutter rate with carotid sinus massage or by administration of AV nodal blocking drugs such as adenosine or verapamil.

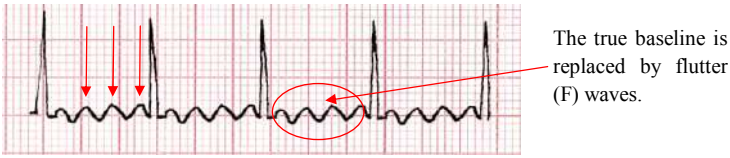


Fig. 4-10 C. The ECG flutter waves are seen as prominent undulation of baseline (saw tooth pattern), the conduction rate 3:1, that is 3-flutter waves for each QRS complex (arrows).

6. Unifocal ectopic atrial tachycardia (paroxysmal atrial tachycardia)

It is tachycardia initiated from ectopic atrial focus rather than SA node by the reentry or automatic mechanism, which represents relatively uncommon type of paroxysmal supraventricular tachycardia (discussed later).

ECG characteristics

1) The P wave morphology differs from sinus P wave, and the R-P interval[1] is longer than P-R interval.
2) The P wave, usually precede the QRS complexes (Fig. 4-11).
3) The direction of P wave either upright or inverted depends on the site of origin, from the upper or lower part of the atria respectively.
4) Ventricular response is usually regular, narrow QRS complex.
5) In case of digitalis intoxication it is associated with the irregular ventricular response, due to frequent block (P waves exceed QRS complexes).
6) Incessant (persistent) form may occur in young patient causing tachycardia-induced cardiomyopathy.

Fig. 4-11. Unifocal atrial tachycardia (a type of paroxysmal supraventricular tachycardia) ends abruptly with resumption of sinus rhythm. The P wave falls on the preceding T wave with prolonged R-P interval (arrow), notice the very rapid rate help to differentiate it from sinus tachycardia and the identification of the baseline between P waves in the sinus rhythm (large arrows) help to differentiate it from atrial flutter with 2:1 AV block.

[1] The long R-P interval is defined as interval more than half of the R-R interval which commonly seen in unifocal atrial tachycardia and rare type of AV nodal re-entrant tachycardia.

Arrhythmias originating in the atrioventricular Junction

In normal situations, the connection between atria and ventricles is called AV junction, which is divided into AV node and bundle of His. Another congenital abnormal connection between atria and ventricles is called accessory pathway that bypass AV node in conduction of the impulse to the ventricles.

Arrhythmias can be classified according to the origin from pathways that connect atria and ventricles, as follows:

1) Arrhythmias with normal conduction pathway

A | AV nodal arrhythmias (AV block discussed later)

B | Bundle of His arrhythmias

2) Arrhythmias with abnormal accessory atrioventricular pathways (e.g., WPW syndrome)

1. Arrhythmias with normal conduction pathway

A | Arrhythmias originating in the AV node

Figure 4-12 illustrates the normal conduction of the impulses through the AV node (alpha and beta pathways).

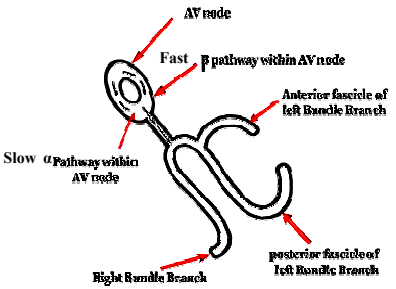


Fig. 4-12. Normal conduction pathways.

AV nodal reentry tachycardia "AVNRT" occurs when there is re-entrant impulses involving one of the two AV node pathways that link the atria to the distal part of the AV node.

- It is the most common form of narrow complex paroxysmal SVT.
- It is more common in female than male, typically manifests in adults and middle aged.
- **The AVNRT is divided into:**
  A | Typical AVNRT (slow-fast)

  It is the most common type of AVNRT, it occurs when the fast (beta) pathway is blocked (Fig. 4-13A), the conduction takes place through slow (alpha) pathway, and the impulse goes back up through the fast pathway to activate the atria at the same time of the ventricular activation (atria and ventricles activated simultaneously), resulting in retrograde P wave buried inside the QRS complex with short RP interval[1].

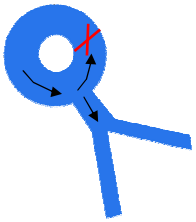


Fig. 4-13 A. Diagram illustrates block of the fast pathway with slow-fast conduction.

Diagnostic ECG manifestation

1) Narrow QRS complex tachycardia.
2) Heart rate ranges from 120 – 250 beats/min.
3) The P wave will frequently be buried inside the QRS complex and it is either not visible or will distort the initial or terminal portion of the QRS complex resulting in pseudo r in V_1 and pseudo s in the inferior leads (Fig. 4-13B).

[1] The short RP interval is defined as RP less than half of R –R interval, which occurs in typical form of the AVNRT and orthodromic AVRT, but in the later the P wave is more clear behind the QRS complex due to long retrograde conduction.

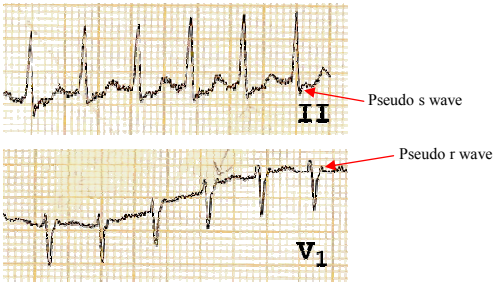


Fig. 4-13 B. AV node re-entrant tachycardia, arrows indicate P wave deforms QRS complex shape resulting in pseudo s and r waves.

B | Atypical (fast-slow) AVNRT

Rarely occur in the clinical practice, when the slow (alpha) pathway is blocked, the conduction is reversed, conducting rapidly down through the fast (beta) pathway and returns up slowly through the alpha pathway. So that the activation of the atria is delayed after the ventricles, thus; the P wave follows the QRS complex with long RP interval (Fig. 4-14A).

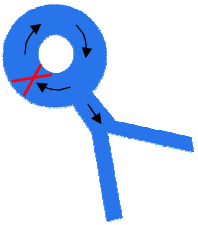


Fig. 4-14 A. Diagram illustrates block of the slow pathway with fast-slow conduction.

Diagnostic ECG manifestation (Fig. 4-14B)

1) Narrow QRS complex tachycardia.
2) Heart rate range from 120 – 250 beats/min.
3) The P wave usually inverted in leads II, III, and aVF, and it clearly follows the QRS complex and usually readily apparent inverted in the preceding T wave with long RP interval.

Fig. 4-14 B. AV node re-entrant tachycardia. The retrograde P waves are clearly seen altering the normal T wave contour (arrow), with prolonged RP interval.

B | Junctional arrhythmias originated from Bundle of His

1. Junctional premature beats

- Inverted P wave may precede, buried, or follow QRS complex.
- QRS complex is usually narrow (unless the conduction is abnormal) and it is followed by compensatory pause (fig. 4-15).
- It may be isolated or multiple (as in bigeminy, trigeminy/ unifocal, or multifocal).

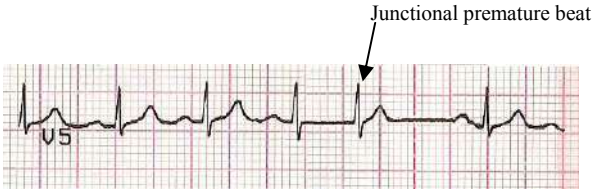


Fig. 4-15. Junctional premature beat, notice the QRS complex without preceding P wave (arrow), followed by compensatory pause.

2. Junctional escape rhythm

- Usually occurs late and not premature when there is SA node arrest (compensatory rhythm).
- P waves, which are often inverted may precede, buried, or follow QRS complex (depending on the origin either high, mid, or low nodal).
- QRS complex is narrow unless there is conduction disturbance.
- Rate is slow (40 – 60 beats/min) (Fig. 4-16).

Fig. 4-16. Junctional escape rhythm, notice the narrow QRS complex not preceded by P wave with heart rate 50 beats/min.

3. Accelerated junctional rhythm

- Rate (60-100 beats/min).
- P wave is inverted, buried in, or follow QRS complex.
- QRS complexes are narrow, unless associated with abnormal conduction (Fig. 4-17).

Fig. 4-17. Accelerated junctional rhythm, notice the narrow QRS complex not preceded by P wave with heart rate 95 beats/min.

4. Junctional tachycardia

- Rate >100 beats/min.
- P wave is usually not visible.
- QRS complexes are narrow, unless there is conduction problem (Fig. 4-18).

Fig. 4-18. Junctional tachycardia, notice the narrow QRS complex not preceded by P wave with heart rate 125 beats/min.

Practical guidelines:

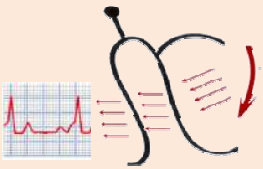
- *Arrhythmias originated from the AV junction are usually caused by enhanced automaticity, abnormal automaticity, or triggered activity, while arrhythmias that originated from AV node are special entity of AV junctional arrhythmias which caused by re-entry mechanism called AV nodal re-entrant tachycardia (AVNRT).*
- *The term upper, mid, or lower junctional rhythm have been replaced recently by the term junctional rhythm.*

2. Arrhythmias with abnormal atrioventricular conduction

Atrioventricular reentrant tachycardia (AVRT) (pre-excitation syndrome)

- Normally the AV node is the only way to conduct the impulses from atria to the ventricles.
- Accessory pathway (bundle of Kent) is abnormally inherited tract conducts the impulses too rapidly from the atria to the ventricles. It bypasses the AV node, resulting in early activation of the ventricles (preexcitation).
- WPW-syndrome accounts the most common causes of preexcitation syndrome, usually manifests in children and young adults; the other rare cause is LGL syndrome is discussed later.

Wolff-Parkinson-white syndrome (WPW-syndrome classically classified into three types)

Type (A) WPW syndrome	*Type (B) WPW syndrome*	*Type (C) WPW syndrome*
It is the most common type of accessory pathway in which: ***1) Impulses travel through the left bundle of Kent.*** ***2) The left ventricle is depolarized first then the septum, and lastly the right ventricle.*** ***3) It produces upright R wave in the right precordial leads (V_1-V_2)*** ***4) ECG manifestation in sinus rhythm:*** • ***Short PR interval.*** • ***Delta wave (Fig. 4-19).*** • ***Wide QRS complex***  V_1-V_2	***1) Impulses travel through the right bundle of Kent.*** ***2) The right ventricle is depolarized firstly, then the septum, and lastly the left ventricle.*** ***3) It produces negative QRS in the right precordial leads (V_1-V_2)*** ***4) ECG manifestation in sinus rhythm:*** • ***Short PR interval.*** • ***Delta wave.*** • ***Wide QRS complex*** V_1 V_2	***Concealed pathway (accessory pathway that conduct only retrogradely)*** ***1) It has been suggested that the accessory pathway may be found in the area of interventricular septum.*** ***2) It is difficult to identify it on ECG in sinus rhythm (no delta wave ,no short P-R interval).***

Practical guidelines:

- *Do not diagnose myocardial infarction, left ventricle hypertrophy, bundle branch block or axis deviation in the setting of the WPW syndrome.*
- *Remember, type A WPW syndrome is one of the differential diagnosis of R wave in V_1 (table 3-2).*
- *Remember, WPW syndrome with sinus rhythm (Fig. 4-19) is an accidental finding at routine ECG, in which the patient does not need further evaluation if he remains asymptomatic, but if symptoms and/or abnormal rhythm appear the patient needs further evaluation.*
- *Remember, the normal ECG without classical manifestation of WPW syndrome does not exclude presence of accessory pathway, which is called in this case concealed accessory pathway.*
- *The 12 lead ECG of WPW syndrome with sinus rhythm provide important clue about the specific location of the bypass tract:*
  - Type A WPW syndrome (the commonest type), the abnormal accessory tract is located in the left ventricle, either in the lateral wall (positive R wave in V_1 and aVF and negative in aVL) (Fig. 4-19), or in the posterior (posterior/septal) wall (positive R wave in V_1 and aVL and negative in aVF).
  - Type B WPW syndrome, the accessory pathway is located anteriorly (anterior/septal) in the right ventricular free wall (negative R wave in V_1 and positive in aVF and aVL) or in the posterior wall (negative R wave in V_1 and aVF and positive in aVL).

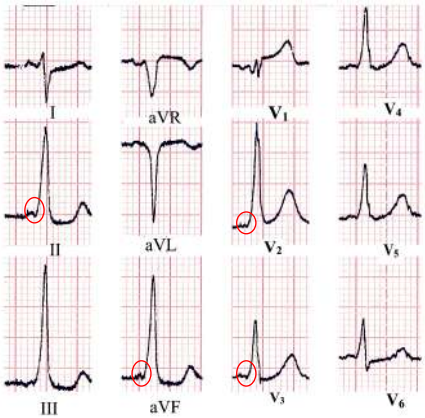
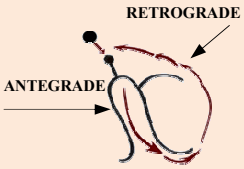
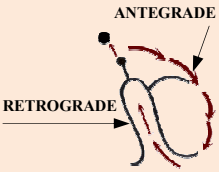
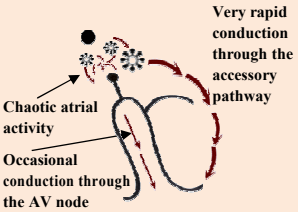


Fig. 4-19. This ECG represents type A WPW syndrome with sinus rhythm .notice the short PR interval less than 3 small squares, slurring of initial portion of QRS complex (delta wave 'circles'), QRS duration 3 small squares, secondary ST –T changes. The classic presentation of WPW syndrome this pattern simulate RBBB, notice the negative delta wave in aVL, and positive delta wave in leads V_1 and aVF probably consistent with a bypass tract inserted in the lateral wall of the LV.

Arrhythmias in WPW-Syndrome

Narrow complex re-entrant orthodromic tachycardia	*Wide complex re-entrant antidromic tachycardia*	*WPW with atrial fibrillation (AF)*
1. It is the most common arrhythmia in WPW syndrome. ***2. The ventricles are activated by the normal AV node.*** ***3. The impulses are conducted antegrade through AV node and retrograde through the accessory pathway.*** *4.* ***ECG manifestation*** *(Fig. 4-20A):* ***- regular, narrow complex tachycardia with the loss of Delta wave(paroxysmal SVT).*** ***- Inverted P wave usually follows the QRS complex, (R-P is shorter than P-R intervals).*** ***- Heart rate usually > 250 beats/min*** ***- Electrical alternans(alternating QRS amplitude)is common.***  	***1. It is rare.*** ***2. The ventricles are activated by the accessory pathway.*** ***3. The impulses are conducted antegrade through accessory pathway and retrograde through the AV node.*** ***4. ECG manifestation*** *(Fig. 4-20B):* ***wide QRS complexes tachycardia***  	***1. It is the second most common and the most serious arrhythmia associated with WPW syndrome.*** ***2. The presence of accessory pathway in patients with atrial fibrillation allows very rapid conduction with fast ventricular response*** ***3. ECG manifestation*** *(Fig. 4-20C):* ***Rapid, completely irregular, broad complex tachycardia but with occasional narrow complex.*** ***Key point:*** ***When wide complex tachycardia becomes narrow consider WPW syndrome with atrial fibrillation which distinguishes it from other causes of wide complex tachycardia .*** 

Note:

In orthodromic activation of the atria through the accessory pathway by atrial premature beat is referred to as an echo-beat.

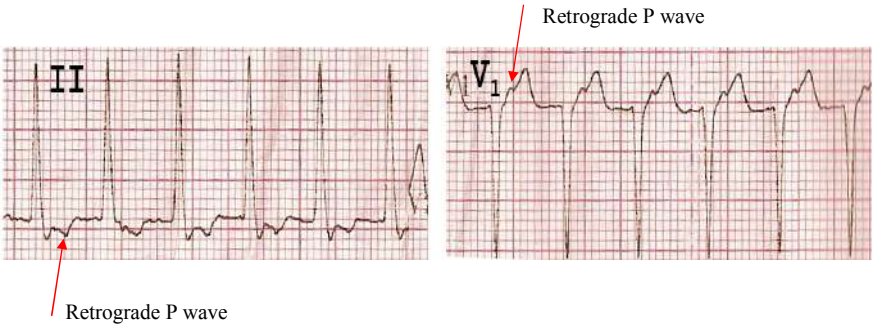


Fig. 4-20 A. Orthodromic narrow complex tachycardia, arrows shows the presence of the retrograde P wave that lies on the ST segment between the QRS complex and T wave with short RP interval (less than half RR interval).

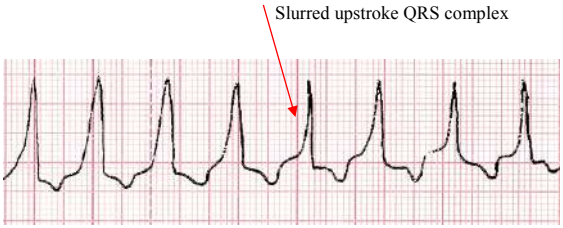


Fig. 4-20 B. Antidromic wide complex tachycardia, the ventricles are completely activated antegradely from the accessory pathway resulting in wide complex relatively regular wide complex tachycardia with slurred upstroke of QRS complex (arrow).

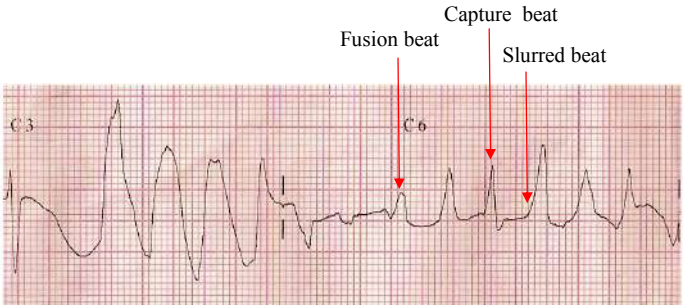


Fig. 4-20 C. The WPW syndrome and atrial fibrillation, the key feature is variably wide QRS at irregular intervals. In the setting of wide QRS complex tachycardia, when QRS becomes narrow consider WPW with atrial fibrillation (capture beat).

Lown-Ganong-Levine syndrome (LGL)

In LGL syndrome, the accessory pathway is the extension of the normal internodal conduction pathway between the atria and bundle of His, and then the conduction to the ventricles from bundle of His is normal (Fig. 4-21A).

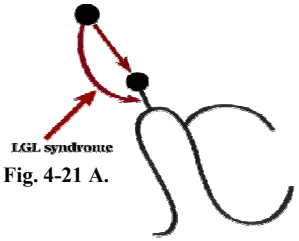


Fig. 4-21 A.

ECG characteristics (Fig. 4-21B)

- Normal QRS complexes.
- No delta wave.
- Short PR interval.

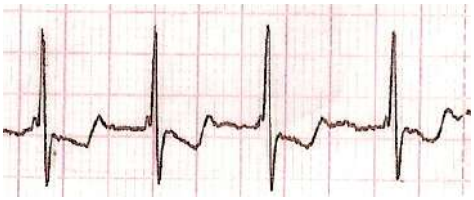


Fig. 4-21 B. LGL syndrome, notice the narrow QRS complex and short P-R interval without delta wave.

Note:

A relatively short PR interval may be seen as a normal variant, without a bypass tract because of accelerated AV conduction; therefore, you should not "overread" an ECG on which the only noteworthy finding is short PR interval.

Paroxysmal Supraventricular Tachycardias (PSVTs)

Do not be confused, Paroxysmal Supraventricular Tachycardias, which are simply a subclass of the supraventricular tachycardia (SVT) but occur in paroxysm (paroxysm means sudden onset). with characteristic ECG manifestation. (Regular narrow complex tachycardia, at average rate 150 to 250 beats/min).

Types of Paroxysmal Supraventricular Tachycardia

Types are based on the identification of P wave:

1) The specific type PSVT (the P wave can be identified) (Fig. 4-22 A), which has three main causes.
   A | AV nodal re-entrant tachycardia (the most common).
   B | Unifocal ectopic tachycardia
   C | AV re-entery tachycardia (orthodromic)

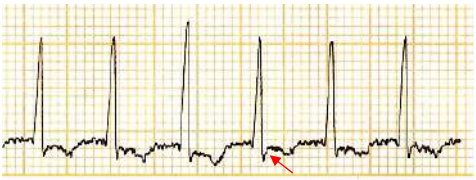


Fig. 4-22 A. Specific type paroxysmal supraventricular tachycardia, the rhythm is regular narrow QRS complex tachycardia, the differentiation between three main causes is made based on the presence of P wave, retrograde P wave are seen between the QRS complex and T wave (arrow), common finding in PSVT is orthodromic type.

2) Non specific type PSVT (P wave which is extremely difficult to be identified) (Fig. 4-22 B), that has the following causes:

A | PSVT (when difficult to differentiate between the three specific types of PSVT mentioned above).

B | Atrial flutter with fixed 1:1 or 2:1 AV block.

C | On rare occasion the sinus tachycardia with heart rate exceeding 150 beats/min.

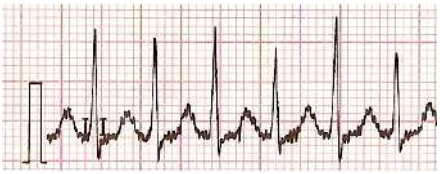


Fig. 4-22 B. Non specific PSVT, notice very rapid tachycardia with narrow QRS complex with no P wave.

Note:

In general practice, if the patient presents with narrow complex tachycardia and non identifiable P wave, do not waste time to search for the type, and treat the arrhythmia as supraventricular origin, then resumption to sinus rhythm will reveal the cause and underlying mechanism, to complete curative and prophylactic treatment.

Atrioventricular (AV) Conduction Block

The atrioventricular conduction block is assessed by examining the relationship between the P waves and QRS complexes as follows:

1) Fixed relationship between P and QRS (first degree AV block).
2) Variable relationship between P and QRS complex (second degree AV block) (Table 4-1).
3) No relation between P and QRS complex (third degree AV block) (Table 4-2).

1. First degree AV block (Fixed relationship between P and QRS)

- Each P wave is followed by QRS complex (Fig. 4-23).
- Fixed prolonged PR interval > 0.2 sec (the conduction is all but constantly delayed).

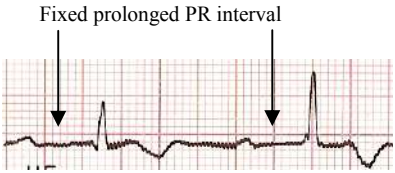
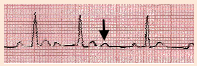
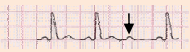
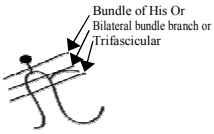


Fig. 4-23. First-degree heart block, notice fixed prolonged P-R interval more than 5 small squares (arrows).

2. Second degree AV block (Variable relationship between P and QRS)

Table 4-1.

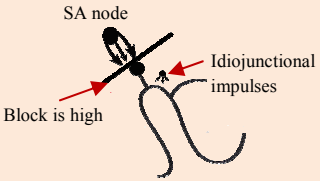
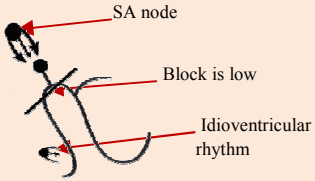
Type of block	Second degree heart block Mobitz type I (Wenckebach)	Second degree heart block Mobitz type II.	Second degree heart block 2:1 advanced block. This block is neither Mobitz I nor II.	High grade second degree heart block
ECG features	Progressive lengthening of PR interval with intermittent dropped beat 	Sudden dropped QRS without prior prolonged PR interval. 	Occurs when every second P wave is conducted to the ventricles.	Occurs when two or more P waves are not conducted
Site	AV node	Infranodal 	Infranodal	Infranodal
	Because it is infranodal and near the bundle branch the QRS is relatively wide. In these types of AV block the conduction is all or none.			
Clinical significant	Usually occurs with inferior MI & has Low risk of progression to complete heart block	High risk of progression to complete heart block		
Therapy	Asymptomatic: no therapy Symptomatic: atropine, may be followed by pacemaker	Temporary pacemaker usually followed by permanent pacemaker	Usually permanent pacemaker	Usually permanent pacemaker
	Secondary causes should be identified and treated, intravenous atropine or catecholamine as a bridge to pacemaker therapy.			

Note :

Ventriculophasic sinus arrhythmia: the PP interval containing the conducted QRS complex is shorter than that, not containing conducted QRS complex.

3. Third degree AV block (complete heart block, no-conduction at all)

Table 4-2.

AV nodal block	Infranodal block
	
1). Atria and ventricles depolarize independently by the SA node and idiojunctional impulses respectively.	**1). Atria and ventricles depolarize independently by the SA node and idioventricular impulses respectively.**
2). The QRS complexes are:	**2). The QRS complexes are:**
A. Less frequent than P waves.	**A. Less frequent than P waves.**
B. Regular R-R intervals.	**B. Regular R-R intervals.**
C. At rate 40-60/min.	**C. At rate 20-40/min**
D. Narrow	**D. Wide**
(P waves > R waves), notice there is no constant relationship between P waves and QRS complexes.	**(P waves > R waves) notice there is no constant relationship between P waves and QRS complexes.**
3). Acute onset, narrow complexes complete heart block may be transient and may respond to atropine.	**3). Wide complex complete heart block is quite dangerous. Dizziness and blackout (Stoke-Adams attacks) often occur, pacing is required.**
4). Chronic narrow complexes heart block may be congenital, then no treatment is required unless being symptomatic; the pacemaker should be applied.	**4). In young usually caused by IHD.**
	5). In elderly usually caused by fibrosis of distal conduction system (Lev's disease).

- Complete heart block can be differentiated from second-degree heart block specially high grade. In the second-degree atrioventricular block, the conducted P wave has fixed relationship with QRS complex.
- Do not forget when treating a patient with complete heart block, secondary causes should be identified and treated.

Atrioventricular dissociation (AV dissociation)

Atrioventricular dissociation simply means no relation between the atria and ventricles. It is usually a secondary manifestation of another rhythm abnormality (not primary diagnosis). It is seen in conditions such as complete heart block, sinus bradycardia or ventricular tachycardia. The diagnosis and treatment are usually directed toward the underlying cause.

AV dissociation is non-specific term and can occur:

1) With conduction disturbance
   - A | AV dissociation caused by complete heart block where there is a total failure of sinus impulse conduction.
   - B | The sinus rate is commonly faster than the junctional or ventricular escape rhythm. In these instances, P waves are more than QRS complexes which is a characteristic finding in complete heart block (table. 4-2.).

2) without conduction disturbance
   - A | Interference AV dissociation, this occurs when the faster junctional or ventricular rate interfere (impede) with the sinus rhythm, e.g., ventricular tachycardia or junctional tachycardia, in these instances P waves are less than QRS complexes this means that the sinus impulse does not prevented completely from passing to the ventricles. The P wave usually is seen marching through the QRS complex or as capture or fusion beats (Fig. 4-24 A).

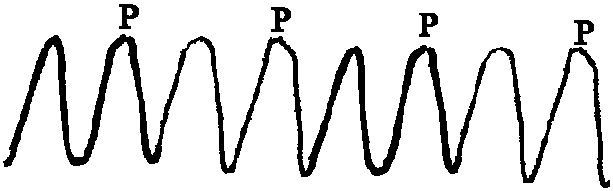


Fig. 4-24 A. Ventricular tachycardia with (AV) dissociation. Notice the dissociation between the P wave and QRS complexes (the numbers of P waves are less than QRS complexes).

B | Isorhythmic AV dissociation, in which the atria and ventricles beating at nearly the same rate, it is usually physiological, e.g., sinus bradycardia when the sinus rate is the same as junctional or ventricular rate (Fig. 4-24 B).

Fig. 4-24 B. AV dissociation isorhythmic type. Notice the type of the rhythm is junctional that occurs during the sinus bradycardia in which the sinus rate is about the same as the junctional rate; however, no relation between P and QRS, the P waves appear to "hid" in the QRS (arrows).

Note:

Every complete heart block represents an atrioventricular dissociation (AV dissociation) but not every AV dissociation represents complete heart block.

Arrhythmias originating in the ventricles
(ventricular dysarrhythmias)

1. Ventricular escape Rhythm (Idioventricular rhythm "IVR")

- The prefix *idio-* indicates the ability for self-initiating of the impulse.
- Ventricular myocardium assumes the pacemaker role, as the primary pacemaker fails or the conduction between atria and ventricles is blocked. The resultant ventricular escape rhythm is usually at the rate of 30-40 beats/min.
- **ECG Characteristics** (Fig. 4-25)
  - P waves are absent or not related to the QRS complexes.
  - Wide QRS complexes and ST-T changes that are directed opposite to the QRS complex.

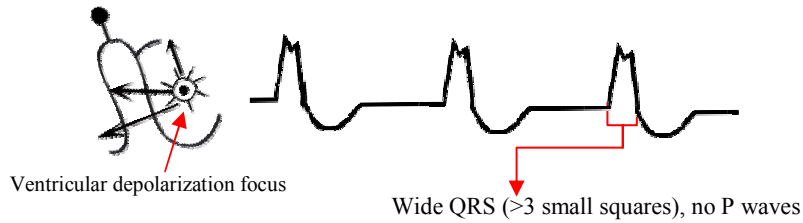


Fig. 4-25. Ventricular depolarization focus at the apex of the left ventricle resulting in wide QRS complex (arrows), without preceding P wave.

2. Accelerated Idioventricular Rhythm (AIVR)

It is the same as IVR, but with relatively increased ventricular rate, but not more than 120 beats/min) (Fig. 4-26 A).

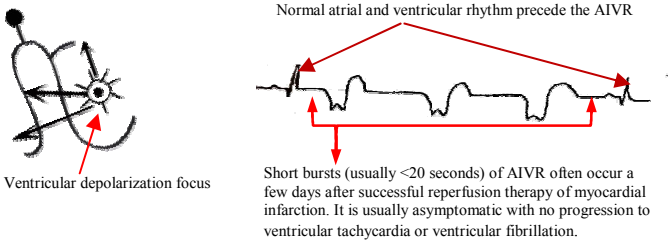


Fig. 4-26 A. Ventricular depolarization focus at the apex of the left ventricle resulting in wide QRS complex (arrows), without preceding P wave, but with increasing ventricular rate.

ECG characteristics (Fig. 4-26B)

- Wide and regular QRS complexes (> 0.10 sec).
- Absence of P wave.
- The rate is usually not more than 120 beats/min

Fig. 4-26 B. Accelerated idioventricular rhythm, notice the wide QRS complex usually is not preceded by P wave, heart rate 85 beats/min.

Practical guideline:

Episodic pattern, short duration, and heart rate less than 120 beats/min, help in differentiation of this benign rhythm from ventricular tachycardia, thus this rhythm should not be suppressed by lidocaine.

3. Ventricular Premature beats (VPBs)

- Ventricular premature beats are the most common dysarrhythmias in clinical practice.
- Ventricular premature beats may occur normally or with organic heart disease

ECG characteristic of VPB

1) It occurs early before the next beat is expected, preceded by relatively fixed coupling interval, and followed by compensatory pause (Fig. 4-27A). In clinical practice, to avoid the confusion between the ectopic and escape beat, some differences are summarized in Table 4-3.
2) QRS duration usually >0.12 second, and ST-T changes that are directed opposite to the QRS complex (Fig.4-27 A).

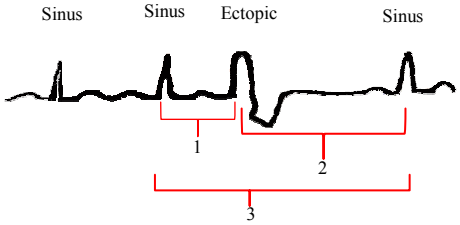


Fig. 4-27 A.
Notice the wide QRS complex of the ectopic beat with ST-T change opposite to the QRS direction.
1) Coupling interval is applied to the interval between VPBs and the preceding normal beat.
2) Compensatory pause is the interval between VPBs and the next normal beat.
3) Complete compensatory pause is applied to the interval between the two sinus beats that surround the VPBs, which equal twice the normal RR interval (2×RR).

VPBs may appear with varying frequencies and shapes as follows:

A | **Couplet** two consecutive premature beats (Fig. 4-27B).

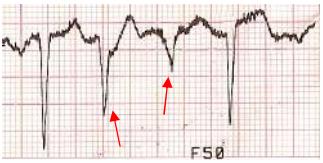


Fig. 4-27 B. Couplet VPBs (arrows)

B | **Bigeminy** every sinus beat is followed by premature beat (Fig. 4-27C).

Fig. 4-27 C. Bigeminy VPBs, every sinus beat (arrow) is followed by premature beat.

C | **Trigeminy** every second sinus beat is followed by a premature beat (Fig. 4-27D).

Fig. 4-27 D. Trigeminy VPBs, notice two sinus beats (arrows) are followed by premature beat.

D | **Quadrigeminy** every third sinus beat is followed by a premature beat (Fig. 4-27E).

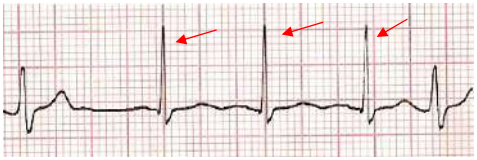


Fig. 4-27 E. Quadrigeminy, notice three sinus beats (arrows) are followed by premature beat.

E | Three or more consecutive premature beats constitute non-sustained ventricular tachycardia (Fig. 4-27F).

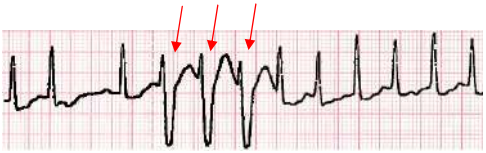


Fig. 4-27 F. Non-sustained VT, notice three consecutive ventricular beats (arrows).

F | **Uniform VPB** it has the same shape in the same lead (Fig. 4-27F).

G | **Multiform VPB** it has a different shape in the same lead (Fig. 4-27G).

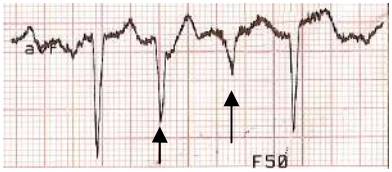


Fig. 4-27 G. Multiform VPBs, the first differs in morphology from the second (arrows).

H | **Interpolated VPB** it is premature beat that occurs between two normal beats, which may not be followed by a pause as usual VPBs (Fig. 4-27H).

Fig. 4-27 H. Interpolated VPB (arrow) without pause.

I | **Multifocal VPBs** premature beats originating from two or more different ventricular locations (Fig. 4-27I).

Fig. 4-27 I. Multifocal VPBs, the upper one originated from right and the lower one from the left ventricles (arrows).

Clinical significance of VPBs

1. In healthy people:

- *Frequently found in normal people, increases with age.*
- *More prominent at rest and tend to disappear with exercise. In contrast to atrial fibrillation which is precipitated by exercise.*
- *The outlook is excellent and the treatment is unnecessary, although low dose beta-blocker sometimes is used to suppress anxiety and palpitation.*
- *Even in healthy subjects, the VPBs may be a manifestation of sub-clinical coronary artery disease. In this regard, no evidence that antiarrhythmic therapy is beneficial in such patients, but the discovery of frequent VPBs might reasonably prompt some general cardiac investigation.*

2. with heart disease:

- *Frequent VPBs in patients who have survived the acute phase of myocardial infarction, the following criteria may indicate poor prognosis:*

  - Multifocal (Fig. 4-27I).

  - Three or more successive beats (non-sustained ventricular tachycardia) (Fig. 4-27F).

  - R on T phenomenon (R wave of the ectopic beat on the T wave of preceding sinus beat) (Fig. 4-28).

- *Unfortunately, antiarrhythmic therapy does not make improvement, and may even worsen the prognosis in that patient.*
- *In patients with heart failure, VPBs are associated with poor prognosis. However, effective treatment of heart failure may suppress the ectopic beats.*
- *VPBs are also a feature of digoxin toxicity.*
- *Sometimes VPBs occur in mitral valve prolapse.*
- *Anyway, the treatment of VPBs is directed toward the underlying cause.*

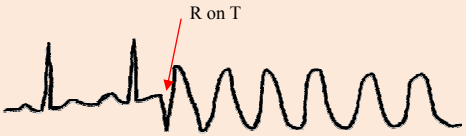


Fig. 4-28. Ventricular ectopic beats interrupting the T wave of preceding beats (R-on-T phenomenon) (arrow). The second such beat initiates ventricular fibrillation.

Table 4-3. Differential diagnosis between

Ectopic and Escape beat

	Ectopic beat	Escape Beat
Mechanism	**Premature Contractions are probably a manifestation of unusual irritability of automatic cells.**	**Occurs as a safety mechanism when the sinus or other dominant rhythm temporarily pauses.**
Onset	**Usually occurs early before the second sinus beat is expected.**	**Occurs late after a pause**
Site of origin	**- Atrial** **- Junctional** **- Ventricular**	**- Atrial** **- Junctional** **- Ventricular**
 Ventricular ectopic beat		 Ventricular escape beat

Note:

A run of three or more consecutive escape beats is named escape rhythm.

4. Parasystole

- Parasystole is a special type of rhythm produced by cardiac cells that function as protected ventricular focus, because it is not penetrated by the sinus impulses. It has the following criteria:
  1) Variable intervals between premature ventricular contraction and the preceding sinus beat (coupling interval) (Figs. 4-29) in contrast to the VPBs, which have fixed coupling interval.
  2) Constant interectopic intervals.
  3) Sometimes fusion beats are noted.
  4) Unchanged QRS configuration of the parasystolic ventricular premature complex (all of them have the same morphology because they are originated from the same focus).

Clinical significance:

- *Parasystole is uncommon arrhythmia, accounts for approximately 3% of VPBs.*
- *Generally, it is benign.*

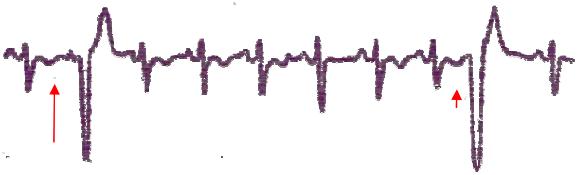


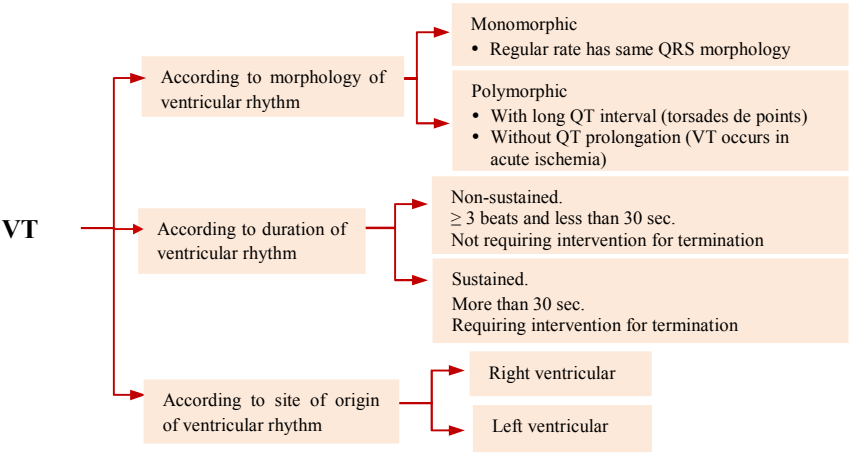
Fig. 4-29. Ventricular parasystole, with variable coupling intervals (long and short arrows).

(Joseph ME. Harrison's Principles of Internal Medicine, 2005, P. 1344)

5. Ventricular Tachycardia

Definition it is three or more successive ventricular premature beats, with rate more than 100 beats/min.

Classification The ventricular tachycardia is generally classified according to the morphology, duration, and site of the origin of ventricular rhythm:



Mechanism commonly re-entry around scared focus caused by ischemia or cardiomyopathy.

Differential diagnosis ventricular tachycardia needs to be distinguished from other causes of wide complex tachycardia such as supraventricular tachycardia with aberrant[1] conduction, or with accessory pathway, i.e., WPW syndrome with pre-excitation (See Fig. 4-20 B of antidromic AVRT)

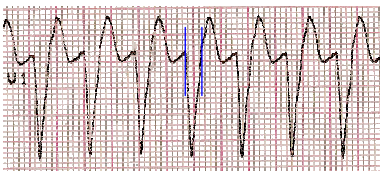


Fig. 4- 30 A. Wide QRS complex tachycardia with LBBB pattern, of uncertain etiology. Full strip ECG, and comparison with ECG of sinus rhythm, are helpful to know the etiology .

[1] Aberrant (abnormal), it is transient conduction abnormality resulting from ability of the impulse to conduct through the less resistant bundle branch (in its relative refractory period), while the other branch is completely resistant to the conduction (in its absolute refractory period) causing transient bundle branch morphology tachycardia.

When doubtful, it is safer to treat wide complex tachycardia as ventricular tachycardia, which is by far the commonest cause of broad QRS complexes, especially in elderly and patients with cardiac disease, because the pre-excitation is less likely to be the first presentation in elderly.

1) Monomorphic ventricular tachycardia

The classic ECG manifestation

It is more likely to be identified when there are:

A | Very wide QRS complexes > 0.14 sec with RBBB or > 0.16 sec with LBBB (the wider the QRS complex, the wider the heart, the more likely ventricular origin of the impulse). It is more helpful if a previous ECG with normal QRS is present for comparison (Fig. 4-30B).

B | Heart rate is greater than 100 beats/min, usually between 150-200 beats/min.

C | Rhythm is usually regular, consistent from beat-to-beat with constant rate.

D | Concordant (same direction either upward or downward) of QRS in all chest leads (V_1 to V_6).

E | Atrioventricular dissociation (Fig. 4-24A).

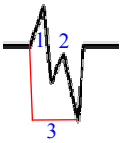
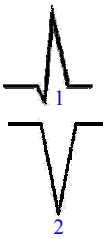
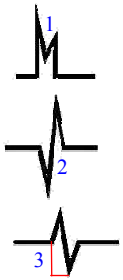
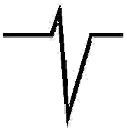
F | Capture beats (Fig. 4-30C).

G | Fusion beats (Fig. 4-30C).

Rarely present but if they are found, the diagnosis of ventricular tachycardia is certain.

H | Extreme axis deviation (between -90° to -180°), i.e., negative complexes in leads I and aVF and positive R wave in lead aVR (because the focus is commonly present at the apex of the ventricles). However, the axis may be normal in a patient with ventricular tachycardia in structurally normal heart (because the focus is present at the base of the ventricles near the origin of the bundle branches such as right ventricular outflow tract tachycardia).

I | QRS morphology in V_1 and V_6

Atypical LBBB		Atypical RBBB	
V_1	V_6	V_1	**V_6**
			
1. wide R> 0.04 sec. **2. Notched S wave.** **3. Onset of QRS to nadir of S> 0.07 sec.**	**The presence of initial Q wave as:** **1. qR or** **2. QS**	**1. Monophasic rabbit ear appearance R wave (the first ear is taller than the second).** **2. Biphasic complex QR.** **3. Biphasic complex RS provide R to nadir S more than 0.10 sec.**	**R/S < 1 (deep S wave).**

Note:

atypical means the QRS not resemble the classic right or left BBB (see chap.5).

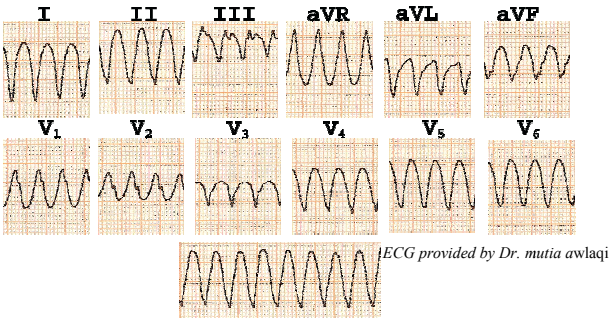


Fig. 4-30 B. Monomorphic ventricular tachycardia. Notice typical wide, regular QRS complexes tachycardia.

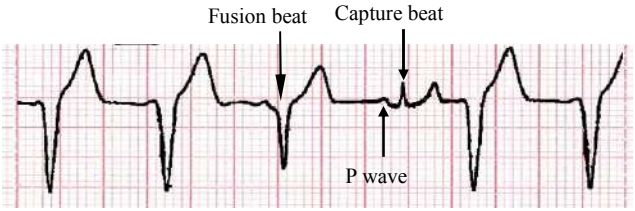


Fig. 4-30 C. Capture beat occurs when atrial impulse stimulates (capture) the ventricles causing normal QRS complex preceded by P wave.
Fusion beat is a normal sinus conducted impulse fuse with an impulse from the ventricular tachycardia.
The ventricles are activated by 2 impulses from the atrial and from the Ectopic focus at the same time. The 2 impulses fuse together produce a beat with deformity appearance that neither looks like the normal beat nor the ectopic ventricular beat called (fusion beat)

2) Polymorphic ventricular tachycardia

A | With prolonged QT interval (Torsades de point)

Characterized by rapid irregular complexes that alternating from upright to an inverted position and seen to twist around the baseline as the mean QRS axis changes in the same lead (Fig. 4-31A).

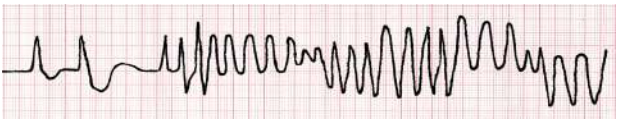
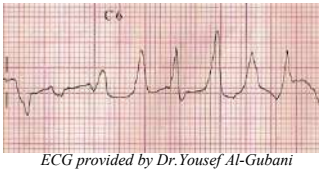


Fig. 4-31 A. Polymorphic ventricular tachycardia, (Torsades de point) and several QRS complexes occur at the peak of preceding T waves R on T that initiate VT, in patients with QT syndrome.
Notice the progressive changes of the QRS amplitude in the same lead which typically occurs in the setting of QT prolongation.

B | Without prolonged QT interval

This type of polymorphic ventricular tachycardia commonly occurs after myocardial ischemia (Fig. 4-31B).



ECG provided by Dr.Yousef Al-Gubani

Fig. 4-31B. Polymorphic ventricular tachycardia without prolongation of QT interval.

Other types of ventricular tachycardia is occasionally seen in clinical practice as follows:

1) Benign ventricular tachycardia
   - Right ventricular outflow tract tachycardia is manifested by left bundle branch block morphology and right axis deviation.
   - Benign left ventricular tachycardia is manifested by right bundle branch block morphology and left axis deviation.

These tachycardias originated from the base of the ventricles near the bundle branch, creating a relatively narrow QRS complex with BBB morphology, usually seen in young adults with normal heart (Fig. 4-30A).

2) Bidirectional ventricular tachycardia, this type of ventricular tachycardia is commonly caused by digitalis toxicity (Fig. 4-31C).

Fig. 4-31C. Bidirectional Ventricular tachycardia with a regular rate and two QRS morphologies with opposite polarities commonly caused by digoxin toxicity.

3) Brugada syndrome, causes ventricular tachycardia, as a first manifestation in some cases and sudden death, the ECG in normal sinus rhythm is a RBBB appearance with coved type ST elevation in $V_1 - V_3$.
4) Arrhythmogenic right ventricular dysplasia (ARDV) see page 60.

6. Ventricular fibrillation (VF)

No clear P waves, QRS complexes, or T waves, the usual ECG manifestation is a fine to coarse deflections (zigzag pattern) (Fig. 4-32).

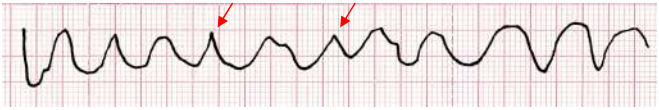


Fig. 4-32. An agonal rhythm is initially present (arrows) but deteriorate into ventricular fibrillation.

7. Ventricular flutter

Very rapid ventricular tachycardia that gives modified pattern on ECG (Regular zigzag) without clearly formed QRS and without definite demarcation between P, QRS, and T waves (Fig. 4-33).

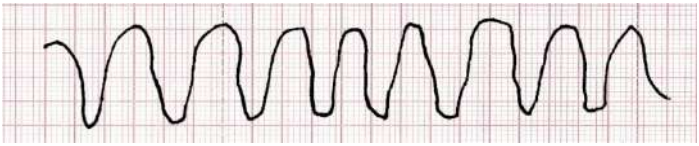


Fig. 4-33. Ventricular flutter

8. Preterminal rhythm

A | Agonal rhythm

It is the occurrence of very broad and irregular ventricular complexes at a slow rate, usually without associated ventricular contraction (Fig. 4-34A).

Fig. 4-34 A. Agonal rhythm

B | A systole

It implies absence of spontaneous cardiac activity and thus, no QRS complexes shown on the ECG (Fig. 4-34B).

Fig. 4-34 B. Asystole; no spontaneous electrical activity and thus no QRS complexes.

Practical approach to diagnose common Tachy/bradyarrhythmias

I) Tachyarrhythmias

1) Narrow complex tachycardia

- Regular:

Sinus tachycardia, paroxysmal SVT and atrial flutter with fixed AV block

- Irregular:

Atrial fibrillation, atrial flutter with variable block and multi-focal atrial tachycardia (any SVT with variable AV conduction).

2) Wide complex tachycardia

- Regular:

Monomorphic ventricular tachycardia, PSVT with preexisting or functional BBB and antidromic WPW syndrome, atrial flutter with fixed AV block and aberrant conduction.

- Irregular:

Polymorphic ventricular tachycardia (torsades de point), atrial fibrillation with WPW or BBB and atrial flutter with variable AV conduction in the setting of BBB or WPW syndrome, MAT with aberrant conduction.

II) Bradyarrhythmias

1) Narrow complex bradyarrhythmia

- Regular:

Sinus bradycardia, first degree heart block, junctional escape rhythm and nodal type third degree AV block.

- Irregular:

Sino-atrial block and second degree AV block.

2) Wide complex bradyarrhythmias

- Regular:

Infranodal type third degree AV block and ventricular escape rhythm.

- Irregular:

Frequent ventricular premature beats with heart rate < 60 beats/min.

III) Abnormal rhythm with normal heart rate

Wandring atrial pacemaker (WAP).

IV) Abnormal rhythm with variable heart rate

Tachy-Brady (Sick sinus) syndrome, AV dissociation and Premature beat (atrial, junctional, or ventricular), sinus arrhythmia.

Treatment of arrhythmias (brief review)

(Do not forget to look for and treat any reversible causes "e.g., electrolyte imbalance specially potassium ").

Brady-arrhythmias

Asymptomatic (hemodynamic stable) do not rush in the treatment if the patient is not symptomatic.

Symptomatic bradyarrhythmias should be treated in the following sequence:

- Atropine 0.5 mg IV in repeated doses every 3-5 min (max 3mg).
- Catecholamines (dopamine, epinephrine, or low dose isopretrenolol).
- Transcutaneous pacing should not be delayed for atropine to take effect.

Tachyarrhythmia

Narrow complex regular tachyarrhythmias

- In haemodynamically, unstable patients synchronized electrical cardioversion DC shock (50J, 100J, 200J, 300J, 360J).
- Haemodynamically stable patient narrow complex, regular usually respond to vagal maneuvers; if not, adenosine IV in repeated doses (6, 12 mg) or verapamil IV (5, 10 mg) slowly, chronic cases usually respond to most anti-arrhythmic drugs and can be cured permanently by catheter ablation.

Narrow complex irregular tachyarrhythmias

Because atrial fibrillation is the most common cause, treatment is by rate control drug (beta-blocker, verapamil and/or digoxin), maintain sinus rhythm (Amiodarone and/or DC shock), and anticoagulant to protect the brain form thrombo-embolism.

If the patient is haemodynamically unstable, the DC shock is the treatment of choice with consideration of anticoagulant.

Wide complex tachycardia

- DC shock[1] is the treatment of choice.
- Intravenous lidocaine, intravenous amiodarone, in refractory cases with normal LV function intravenous procainamide and cardioversion if there is LV dysfunction.

[1] DC is direct current cardioversion electrical shock delivered to the heart through electrodes paddles placed on the chest wall.

- Wide complex tachycardia with variable QRS duration and morphology (e.g., AF and WPW syndrome), should never be treated with AV blocking agents (digoxin, beta-blockers and verapamil).
- For chronic prophylactic therapy (amiodarone or mexiletin), implantable cardiac defibrillator (ICD)[1] is an effective alternative.
- If the cause is torsades de point, it usually responds to magnesium sulphate, overdrive pacing, or isopretrenolol.
- Try to avoid catecholamines (epinephrine, dopamine, dobutamine) as a primary treatment in a patient with tachyarrhythmias.
- If the patient is in the terminal rhythm or asystole, then the empirical treatment should be given as CPR[2], asynchronized DC shock, IV epinephrine, IV atropine, or sodium bicarbonate.

Consult texts, guidelines for further reading.

Notes:

- *Do not combine verapamil and beta-blockers (can cause asystole).*
- *In the treatment of tachy/bradyarrhythmia, remember that the target heart rate should be kept between 60 – 100 beats/min.*
- *Because arrhythmia is considered an emergency in clinical practice, there is no substitute beside clinical history for ECG to diagnose the arrhythmias.*
- *Types of ECG tests used for detection of cardiac arrhythmias are:*

  12-lead ECG, in-hospital ECG monitoring, ambulatory (Holter) ECG monitoring, patient's activated ECG recording, exercise test, and tilt test.

[1] ICD used to deliver electrical shock to the heart through small intracardiac electrodes.
[2] CPR cardiopulmonary resuscitation.

CHAPTER 5

Intraventricular Conduction Defects

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Intraventricular Conduction Defect

Recall the sequence of normal cardiac conduction

The result of normal depolarization of the ventricles is normal QRS in morphology and duration, preceded by normal P wave and PR interval. When QRS differs in morphology and/or duration, with normal atrial P wave and PR interval, this becomes the basic diagnostic criterion for bundle branch block. It may be fixed (at all heart rates) or intermittent (tachycardia or bradycardia dependent), organic or functional.

- Normal activation of the septum is from left to right, produces a small positive septal r wave in V_1 and small negative septal q wave in V_6 (Fig. 5-1A).

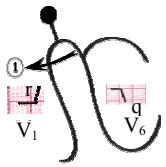


Fig 5-1 A. Arrow 1 represents the normal septal depolarization.

- The left and right ventricles are simultaneously depolarized. The left ventricle is much thicker than the right, so it generates more voltage in the left precordial leads, which produces deep S wave in V_1 and tall R wave in V_6 (Fig. 5-1B).

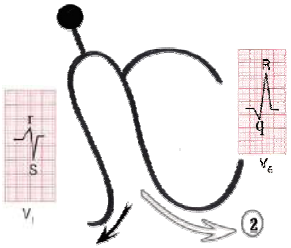


Fig 5-1 B. Ventricular depolarization, the large and small arrows represent the left and right ventricular electrical forces respectively, the left ventricle is responsible for most of the heart forces.

1. Right bundle branch block (RBBB)

A | Complete RBBB

Only the terminal phase of ventricular depolarization is affected by the RBBB, as the following steps:

Step 1: The septum is depolarized from left to right produces positive small septal r wave in V_1 and negative small septal q wave in V_6 (Fig. 5-2A).

Fig. 5-2 A. RBBB, notice the normal depolarization of the septum from left to right (arrow).

***Step 2*:** The left ventricle is depolarized from the right produces S in V_1 and R in V_6 (Fig. 5-2B).

Fig. 5-2 B. RBBB, notice left ventricular depolarization before the right ventricle (arrow).

Step 3: The right ventricle is depolarized slowly from left side (because the right bundle branch is blocked), so, it produces wide R' wave in V_1 and slurred S wave in V_6 (Fig. 5-2C).

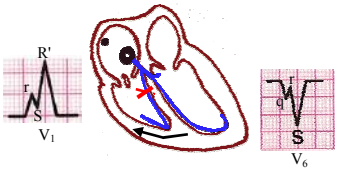
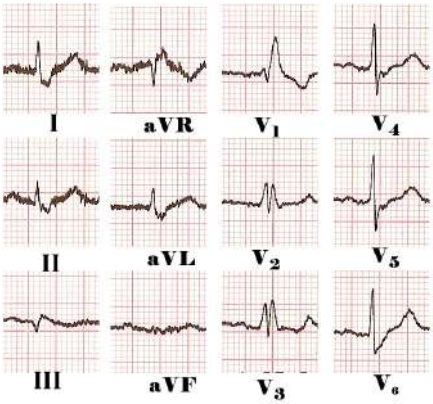


Fig. 5-2 C. RBBB, notice the RV depolarization after the left ventricle (arrow).

Diagnostic ECG criteria for complete RBBB (Fig. 5-2D)**.**

1) QRS duration ≥ 0.12 sec
2) Broad notched R wave in V_1
3) Deep, wide, slurred S wave in V_5-V_6

4) ST segment depression and T wave inversion in (V_1-V_3). The T wave inversion or ST depression in other leads (V_4-V_5) represents a primary change, perhaps resulting from ischemia or drug effect.
5) Normal septal q wave in left precordial leads
6) Delayed R wave peak time (intrinsicoid deflection) in V_1 and V_2 ≥ 50 msec



block (RBBB)

- Wide QRS complex
- rSR' in $V_1 – V_3$
- Deep, slurred S in V_6

B | Incomplete RBBB

Diagnostic ECG criteria for incomplete RBBB are the same as complete RBBB but with QRS complex duration between 0.08 and 0.12 sec (Fig. 5- 2E).

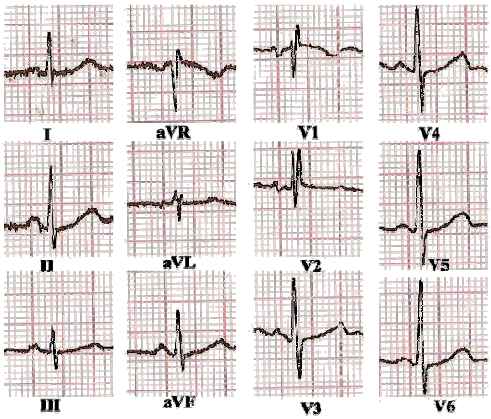


Fig. 5-2 E. Incomplete right bundle branch block

- Morphology of the RBBB.
- QRS duration < 0.12 sec.

Practical guideline:

M-shaped QRS complex in V_1 and W-shaped QRS complex in V_6 may help you to remember the QRS complex of RBBB.

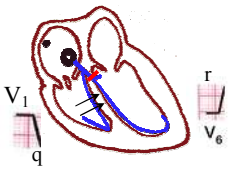
2. Left bundle branch block (LBBB)

A | Complete LBBB

The sequence of ventricular activation is almost different than that which occurs in RBBB, the major reason for this difference is that RBBB affects mainly the terminal phase of ventricular activation, whereas LBBB affects the initial and terminal phases.

- The septum is activated from right to left (reversal of the normal pattern), producing small q in V_1 and r in V_6. Thus, the first major ECG change produced by LBBB is the loss of both normal septal r wave in V_1 and normal septal q wave in V_6 (Fig. 5-3A).

Fig. 5-3 A. LBBB, notice activation of the septum from the right to the left (arrows).



- Activation of thin-walled right ventricle from left to right produces small r wave in V_1 and small s wave in V_6 (Fig. 5-3B).

Fig. 5-3 B. LBBB, notice the right ventricle activation before the LV (arrow).

- The delayed depolarization of thick-walled LV from right shifts the electrical force toward the left produces deep S in V_1 and tall R wave in V_6 (Fig. 5-3C).

Fig. 5-3 C. LBBB, notice the delayed activation of the LV from the right (arrow).

Diagnostic ECG criteria of Complete LBBB (Fig. 5-3D)

1) QRS duration ≥ 0.12 sec.
2) Broad slurred or notched R wave in lateral precordial leads V_5, V_6, lead I and aVL without a preceding Q wave. V_6
3) ST-T changes that are directed opposite to the direction of QRS complex.
4) QS complex in leads V_1-V_2 (in rare cases, complete LBBB appears as a wide rS complex in V_1). V_1
5) Delayed R wave peak time (intrinsicoid deflection) in V_5 - V_6 ≥ 60 msec.

Practical guidelines:

- *W-shaped QRS complex in V_1 and M-shaped QRS complex in V_6 may help you to remember the QRS complex of LBBB.*
- *Occasionally step 1 and step 2 may not appear in the surface ECG and only step 3 resulting in wide deep S wave (QS) in lead V_1 and wide tall monophasic R wave in lead V_6 (Fig. 5-3D).*

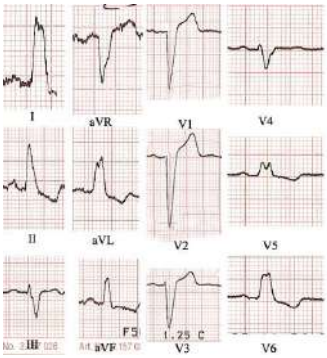


Fig. 5-3 D. Complete Left bundle branch block
Essential features:

- Broad QRS complexes.
- QS in V_1, V_2, V_3
- Notched R wave in V_5, V_6

Incomplete LBBB

Diagnostic ECG criteria of incomplete LBBB have the same pattern of complete LBBB, but with QRS duration between 0.08 and 0.12 sec (Fig. 5-3E).

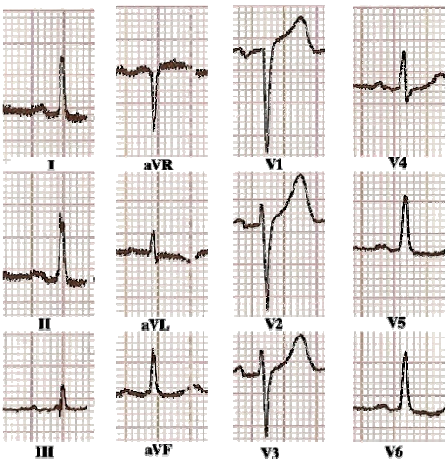


Fig. 5-3 E. Incomplete left bundle branch block
Essential points:

- LBBB morphology.
- Normal QRS duration.

3. Fascicular hemiblock

Anatomical consideration

- The anterior (anterosuperior) division of left bundle branch is more vulnerable to interruption than the posterior (posteroinferior) division, due to the following reasons:

  - The anterior division is long and thin, whereas the posterior is relatively short and thick.

  - The posterior division has double blood supply, in contrast to anterior, which has a single blood supply by septal branch of anterior descending artery, which also supplies the right bundle branch. This is one of the reasons of frequent manifestations of RBBB with left anterior hemiblock.

  - The anterior division is closer to the aortic valve; therefore, it is more likely to be involved in disease affecting the aortic valve.

A | Left anterior (anterosuperior) hemiblock

Figure 5-4 A. illustrates the mechanism of left anterior hemiblock. Shift of the electrical vector to the left causing marked left axis deviation without significant widening of QRS complex (qR in lead I, aVL and rS in the inferior leads).

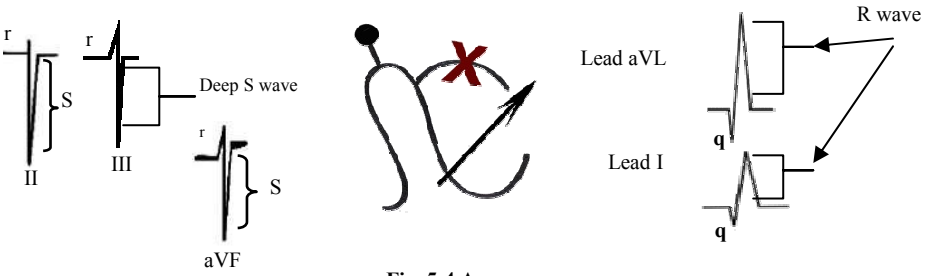


Fig. 5-4 A.

Diagnostic ECG criteria of left anterior hemiblock (Fig. 5-4B)

- Marked left axis deviation LAD (- 45º to -90º)

  i.e., qR in lead I, aVL

  rS in inferior leads II, III, aVF

- Normal QRS duration and morphology.
- Exclude other causes of LAD, e.g., LVH, WPW type B, inferior MI.

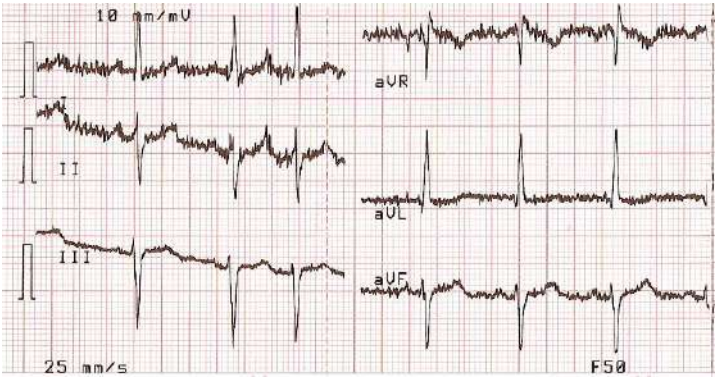


Fig. 5-4 B. left anterior hemiblock. Notice qR in leads I and aVL and rS in leads II, III, and aVF. marked LAD.

B | Left posterior (posteroinferior) hemiblock

Figure 5-5 A. illustrates the mechanism of left posterior hemiblock. Shift of the electrical vector to the right causing marked right axis deviation without significant widening of QRS complex (rS in lead I, aVL and qR in inferior leads II, III, aVF).

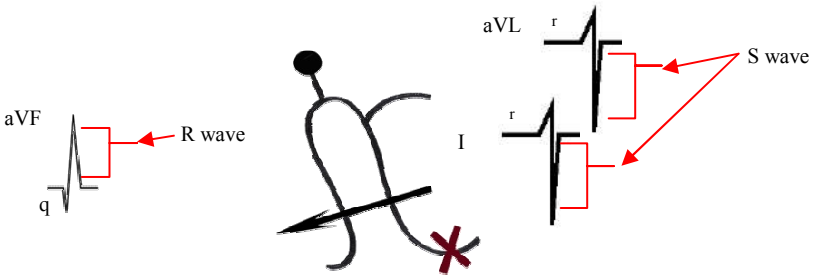


Fig. 5-5 A.

Diagnostic ECG criteria (the opposite of left anterior hemiblock) (Fig. 5-5B)

- Marked right axis deviation (RAD) (+120° to +180°).

  i.e., rS in lead I, aVL.

  qR in inferior leads II, III, aVF

- Normal QRS duration and morphology.
- Exclude other causes of RAD e.g., RVH, anterolateral MI, WPW type A.

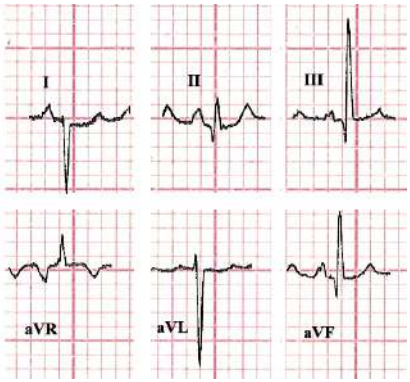


Fig. 5-5 B. left posterior hemiblock.
Notice:
qR in leads II, III, and aVF.
rS in leads I and aVL.
Marked RAD.
Normal QRS complex duration.

C | Bifascicular block

1) It is right bundle branch block with one of the following:
   - Left anterior hemiblock (LAH) produces a RBBB pattern with marked LAD (common) (Figs. 5-6 A and C).

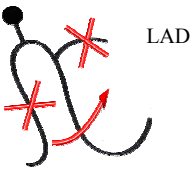


Fig. 5-6 A. Bifascicular, notice the direction of electrical current causing LAD (arrow) in case of left anterior hemiblock with RBBB.

- Left posterior hemiblock (LPH) produces a RBBB pattern with marked right axis deviation (RAD) (Fig. 5-6B).

Fig. 5-6 B. Bifascicular, notice the direction of electrical current causing RAD (arrow) in case of left posterior hemiblock with RBBB.

2) Complete LBBB, which indicates blockage of both anterior and posterior fascicles.

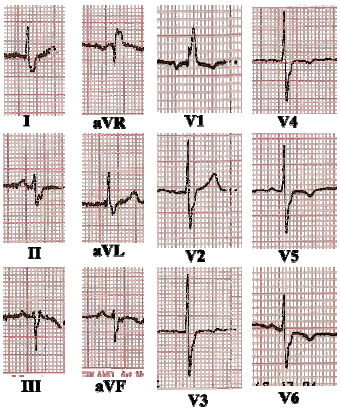
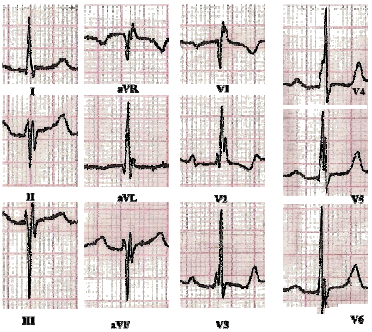


Fig. 5-6 C. Bifascicular Block
Essential features:
- Right Bundle Branch Block
- Left anterior hemiblock with marked LAD -60°

D | Trifascicular block

It is a bifascicular block with prolonged PR interval (Fig. 5-7).

Fig. 5-7. Trifascicular Block
Essential features:
- Right Bundle Branch Block.
- Left anterior hemiblock with marked LAD -80°).
- First degree AV Block.



Other variants of bundle branch block

A | Alternating bundle branch block

1) Conduction abnormality involving both the right and left bundle branches.
2) Indicated by the presence of some alternating beats with RBBB and other with LBBB or by AV block located distal to the common bundle (Fig. 5-8A).

Fig. 5-8A. Alternating BBB, notice the RBBB (short arrow) and LBBB (long arrow), notice the prolonged R-R interval indicates associated AV block which carry poor prognosis.

B | Intermittent (rate dependent) aberrant intraventricular conduction

When one bundle branch is unable to conduct the impulse temporarily usually RBB, the impulse will be conducted

through the LBB, causing aberrant conduction with RBBB morphology, also LBBB morphology may occur when the impulse conduction occur down the RBB (Fig. 5-8B).

Commonly appears when the heart rate exceeding or decreasing than certain critical value (tachycardia or bradycardia dependent).

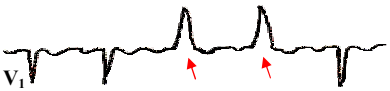


Fig. 5-8B. Intermittent BBB (rate dependent), notice the wide QRS complex with RBBB morphology (arrows) in V_1,because the RV is activated transiently from left bundle branch.

C | Non-specific intraventricular conduction defect

1) Widening of QRS complex with morphology not consistent with one of bundle branch block (either left or right), or fascicular block (Fig. 5-8C).

2) Widening of the QRS complex with morphological combination of both left and right bundle branch block in the same ECG.

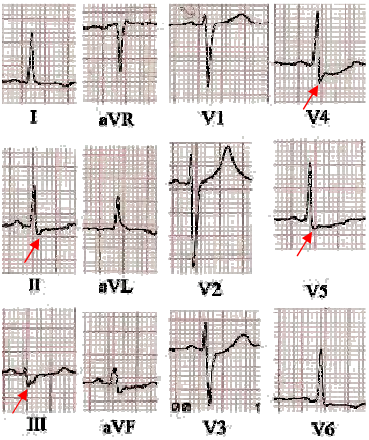


Fig. 5-8 C. Non-specific intraventricular conduction defect, notice wide QRS without a typical features of RBBB or LBBB morphology. Notice also the QRS complexes with terminal delay (arrows).

Other terms used in non-specific intraventricular conduction disturbance:

- Pre-infarction block, transient widening of the QRS complex in the presence of Q wave, the terminal portion of the QRS complex is directed opposite to the Q wave in inferior or lateral leads.
- Pre-ischemic block, transient widening of the QRS complex in the presence of ST deviation, seen in acute injury.

Practical guidelines:

- *The diagnosis of the left anterior or left posterior hemiblock is inferred from marked deviation of QRS axis without changes in QRS morphology or duration (After exclusion of other causes of axis deviation). Be careful, in clinical practice, the fascicular block is more specific diagnosis than axis deviation.*
- *The diagnosis of LBBB and RBBB is inferred from the QRS pattern (morphology and duration) without change of the mean QRS axis, and if there is associated axis deviation, this indicates the presence of other cardiac pathology e.g., RBBB with LAD indicate ASD primum type* (Fig. 3-10), *complete LBBB with LAD associated with myocardial infarction indicate poor prognosis due to more muscle damage* (Fig. 5-9).
- *The diagnosis of ventricular hypertrophy in the presence of bundle branch block is difficult in clinical practice. A few general guidelines are helpful:*

  - The diagnosis of LVH in the setting of LBBB is suggested by the presence of left atrial enlargement and left axis deviation.

  - The diagnosis of RVH in the setting of RBBB is suggested by the presence of right atrial enlargement, tall R wave in V1 ≥ 15 mm, and right axis deviation.

- *The ECG diagnosis of MI in the presence of bundle branch block is discussed in ischemic heart disease (chapter 6).*
- *In case of LBBB, the left precordial leads V_5, V_6, lead I and aVL cannot reflect septal q wave or terminal S wave.*
- *LBBB may be the first clue to previously undiagnosed but clinically important abnormalities, these are advanced coronary artery disease, valvular heart disease, hypertensive heart disease, and cardiomyopathy.*
- *The commonest cause of left axis deviation (LAD) is the left anterior hemiblock due to fibrosis that may result from IHD, chronic heart failure, chronic hypertension, and cardiomyopathy.*
- *RBBB is generally of minor clinical significance, except if associated with left or right axis deviation may indicate heart disease.*

- *Alternating RBBB and LBBB of new onset in the setting of MI carry high risk of progression to complete heart block, and permanent pacemaker is required.*
- *Deep S wave in left precordial leads is a manifestation of RVH and RBBB but the presence of slurring S wave is more characteristic of RBBB than RVH.*

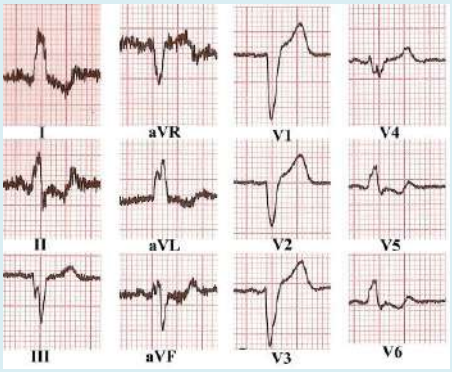


Fig. 5-9. LBBB and left axis deviation

- *The student and general practitioner must understand that wide QRS complex is not restricted to BBB. Other causes of wide complex should be considered such as:*

  - Right or Left Bundle Branch Block.

  - Toxic conduction delay caused by hyperkalaemia or certain drugs.

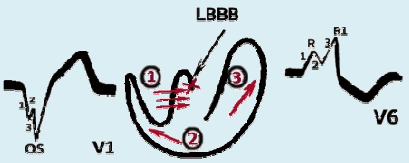
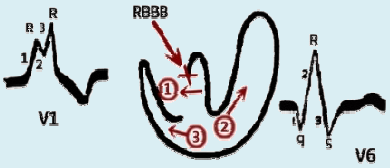
  - Ventricular tachycardia (Fig. 4-30B).

  - Pacemaker beats (Fig. 8-4B).

  - WPW syndrome (Fig. 4-20C).

- *The pure BBB by itself does not need treatment, but keep in mind in general practice, it is essential to scan all ECG leads to see the coexisting axis deviations, AV block, and ST-T changes, if present the prognosis and the treatment are significantly changed.*

Table 5-1 Summary of pathophysiology and ECG changes of Bundle Branch Block

Complete Left Bundle Branch Block (LBBB)	Complete Right Bundle Branch Block (RBBB)
	
1) The septal activation occurs from right to left (a reversal of the normal pattern) produces Q wave in V_1 and R wave in V_6 (arrow 1 above) (there is no positive septal r wave in V_1 and no negative septal q wave in V_6). 2) Depolarization of right ventricle normally produces r wave in V_1 and S wave in V_6 (arrow 2 above). 3) Delay depolarization of the left ventricle due to LBBB, produces S wave in V_1 and R wave in V_6. Therefore, the QRS complex is wider than 3 small squares with abnormal shape (arrow 3 above).	1) Septum depolarization occurs from left to right (same as the normal pattern) (arrow 1 above) produces small septal r wave in V_1, and small septal q wave in V_6. 2) Depolarization of left ventricle normally produces S wave in V_1, and R wave in V_6 (arrow 2 above). 3) Late depolarization of right ventricle from left, produces R wave in V_1 and S wave in V_6 (arrow 3 above). Therefore, the QRS is wider than 3 small squares with abnormal morphology.

CHAPTER 6

Myocardial Ischemia and Infarction

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Ischemic Heart Disease (IHD)

Introduction

- IHD remains the leading cause of morbidity and mortality in the world.
- Coronary artery disease (CAD) represents stable angina (angina pectoris), acute coronary syndrome, heart failure (HF), silent ischemia, and sudden death.
- Acute coronary syndrome represents a spectrum of clinical presentation of acute myocardial ischemia referred to as unstable angina (UA), non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).
- From the clinical point of view, the division of acute myocardial infarction into ST-segment elevation and non-ST-segment elevation type is useful since the efficacy of acute reperfusion therapy is limited to the former group.
- The ECG is considered the single most important initial clinical test for diagnosis of IHD in patients with chest pain, but before you comment on ECG changes, there are many considerations to account:
  - The ECG has important limitations in both sensitivity and specificity in the diagnosis of IHD.
  - A single normal ECG does not exclude ischemia or acute MI.
  - A prolonged chest pain without diagnostic changes in serial ECGs makes acute coronary syndrome less likely etiology of chest pain.
  - Other conditions that may mimic ECG manifestations of myocardial infarction are summarized in Table 6-1.

Table 6-1. *Some cardiac and non-cardiac conditions that can mimic myocardial infarction*

Diagnosis	ECG findings mimicking MI	Diagnostic evaluation
Pericarditis	ST elevation	Echocardiography
Myocarditis	ST elevation, Q wave	Echocardiography
Acute aortic dissection	ST elevation or depression, non specific ST-and-T-wave changes	Transesophageal echocardiography, CT of the chest, aortography or MRI
Pneumothorax	New poor R-wave progression in leads V_1-V_6, and acute shift of QRS axis	Chest radiography
Pulmonary embolism	Inferior ST elevation, ST shift in lead V_1-V_3, and sinus tachycardia	Ventilation perfusion scan
Acute cholecystitis	Inferior ST-elevation	Abdominal ultrasound or radioisotope scan

Source: Smith, S.C. and Goldberg, AC., The Washington Manual of Medical Therapeutic, 2001,p.106

- The diagnostic ECG changes of ischemia and/or infarction are often masked by the presence of the following conditions:
  1) Left bundle branch block.
  2) Electronic ventricular pacemaker.
  3) WPW syndrome with Preexcitation.
  4) Chronic digoxin use.
  5) Left ventricular hypertrophy.

Thus, the clinical judgment, previous ECG and biomarkers are useful to confirm the diagnosis.

- The most important aspects to consider in ischemia and MI are:
  1) ECG changes are limited to the area which is affected by specific coronary artery (respect anatomical distribution).
  2) Reciprocal changes means ECG changes seen in leads overlying the opposite wall of the infraction site.
- Generally, pathological changes in IHD (Fig. 6-1) and ECG manifestations are described below:

  A. Ischemia.

  B. Injury.

  C. Necrosis.

A | Ischemia causes transient ST segment depression and/or elevation (prinzmetal's angina), or symmetrical T wave inversion.

B | Injury causes ST segment elevation and/or depression with or without loss of R wave progression, with more prolonged ECG changes than ischemia.

C | Necrosis (infarction) causes permanent deep Q wave (because of the absence of depolarization), and deep T wave inversion.

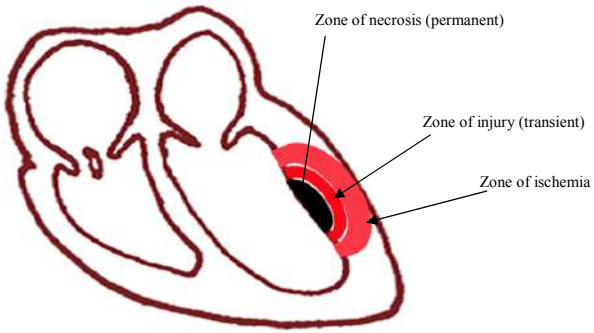


Fig. 6-1. Notice the progression of pathological changes of myocardial ischemia/infarction.

Angina Pectoris

It is chest discomfort results from myocardial ischemia (angina means pain or discomfort) which is generally divided into three types:

1) Chronic stable angina

It is typical chest discomfort usually occurs at exertion and relieved by rest.

Diagnostic ECG changes

The diagnostic evaluation of suspected stable angina is guided by the estimated pretest probability that a patient has a coronary artery disease.

A | Because of normal ECG between the attacks in relation to the stable angina itself, other tests are considered for the diagnosis e.g., treadmill exercise test. The brief practical points of treadmill test are summarized below (Fig. 6-2 A and B).

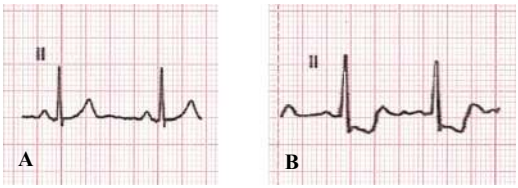


Fig. 6-2. In patient with stable angina.
A. Normal ECG resting strip in a patient with coronary artery disease.
B. Notice the horizontal ST segment depression after the end of treadmill exercise test, Indicating myocardial ischemia.

B | During the attack, the most common ECG changes include non-specific ST-T changes (horizontal or downsloping ST segment depression and/or symmetrical T wave inversion, T wave pseudonormalization[1]) in two or more contagious leads (Fig. 6-3 A and B).

Fig. 6-3.
A. ECG strip obtained from patient during anginal pain, there is ST segment depression.
B. after administration of sublingual nitroglycerin and subsequent resolution of angina.

2) Unstable angina

Rapidly progressive angina, new onset angina, angina after MI ECG changes are the same as stable angina, but lasts longer and usually not related to exercise. Unstable angina is differentiated from acute non-ST elevation myocardial infarction by biomarkers, although, it may be elevated but not to the same extent of myocardial infarction. Thus patient history, serial biomarkers and ECG monitoring are essential.

3) Variant angina (prinzmetal's angina)

Typically, unpredictable transient chest pain often occurs at rest, usually at early morning.

[1] Pseudonormalization ECG with already inverted T-wave that becomes upright during chest pain.

ECG changes

Transient ST-segment elevation during the attack. As the patient is rarely seen with spontaneous attack, it is necessary to carry out the continuous ECG monitoring test[1] for the diagnosis (Fig. 6-4 A and B).

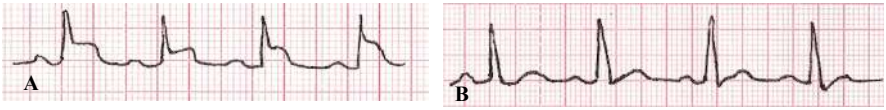


Fig. 6-4.
A. ECG monitoring test in a patient with chest pain demonstrates transient ST-segment elevation during the attack.
B. ECG strip several minutes after spontaneous resolution of pain shows normal ECG.

Notes:

- *Ischemic ECG changes, without anginal chest pain is called silent ischemia.*
- *Atypical presentation of angina such as angina presents as dyspnoea is called angina equivalent. When angina occurs on lying down it is called decubitus angina. When angina occurs at night (and may awake the patient from sleep) is called nocturnal angina.*

Brief practical pointes of treadmill test:

- *Exercise treadmill test depends on the pretest probability of coronary artery disease (e.g., risk factors for coronary artery disease).*
- *ST segment depression (horizontal or downsloping) at least 1mm or more, lasting 0.08 sec (2 small square) is considered positive test. ST-segment elevation in leads without previous Q wave of old MI indicates sever coronary artery disease.*
- *Patient unable to perform the exercise, or patient with secondary ST-T changes should not be referred for exercise treadmill test, because the difficulties in the interpretation such as, LVH, BBB, WPW syndrome, on digoxin use, ventricular pacing, or previous CABG.*
- *Normal (negative) exercise ECG test does not exclude coronary artery disease.*
- *Stress test should be performed in absence of anti-anginals agents.*

[1] Continuous ECG monitoring test is an ECG recording of one or more leads, either for the detection of abnormalities in morphology suggestive of ischemia or rhythm abnormalities.

Myocardial infarction

The diagnostic ECG changes of MI should be correlated strictly with clinical presentation and cardiac biomarkers.

Always remember, the access to the patient's previous ECG assists in accuracy of interpretation and detection of new abnormalities.

While reading an ECG of MI, the following points should be considered:

A | Type of infarction.
B | Age of infarction.
C | Site of infarction.

The extent of myocardial infarction and ossociated other ECG Abnormalities must be considerd.

A. Types of infarction

1) ST-segment elevation myocardial infarction(STEMI) (Fig. 6-5A)

It is a new ST elevation at J point ≥ 2 mm in two or more contagious chest leads or ≥ 1 mm in two or more contagious (adjacent) limb leads.

2) Non-ST-segment elevation myocardial infarction(NSTEMI) (Fig.6-5B)

It is a new horizontal or down sloping ST segment depression ≥ 0.5mm and/or symmetrical T wave inversion ≥ 2mm with prominent R wave[1] in two or more contagious leads. On clinical ground, symmetrical T wave inversion ≥ 2mm is strongly suggetive of acute ischemia.

[1] The prominent R wave helps us to differentiate T wave inversion of acute MI from that of post-infarction T wave inversion.

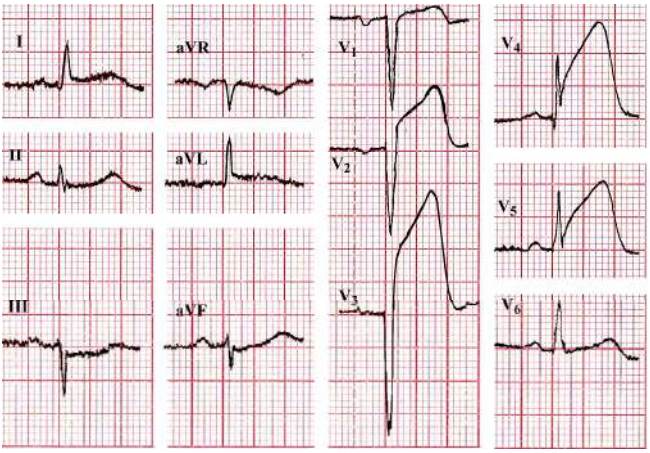


Fig. 6-5 A. ST segment elevation MI, notice ST segment elevation in V_2 - V_5 with poor R wave progression, and pathological Q wave in V_1 – V_4.

Fig. 6-5 B. Non-ST elevation MI, notice the deep T wave inversion in leads V_2-V_6, obtained from a patient with chest pain and elevated cardiac biomarkers.

B. Age of infarction

1) Acute myocardial infarction

Characterized by the following ECG changes (Fig. 6-6)

A | New ST-segment elevation, with or without Q wave (STEMI).

B | New ST depression and/or T wave inversion (NSTEMI).

Supportive changes :

- Hyperacute T wave at least in two contagious leads (it is an early sign that may precede ST segment elevation).
- Reciprocal changes (ST depression in leads opposite to the infarction site).

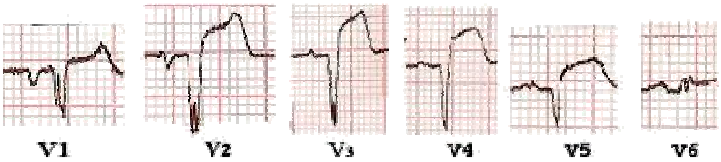


Fig. 6-6. ECG of acute anterior myocardial infarction taken during first 24 hours, notice the ST elevation and Q wave in leads $V_1 – V_6$.

2) Recent infarction

It is an infarction aged more than 7 days and less than 30 days with variable ECG changes as follows:

- Isoelectric ST segment.
- Deep Q waves and inverted T waves (Fig. 6-7).

Fig. 6-7. Recent MI : notice Isoelectric ST segments and deep symetrical T waves inversion.

3) Old infarction (age indeterminate)

More than one month age, with the following ECG features (Fig. 6-8):

- Isoelectric ST segment .
- Deep Q wave and normal or non specific T wave inversion.

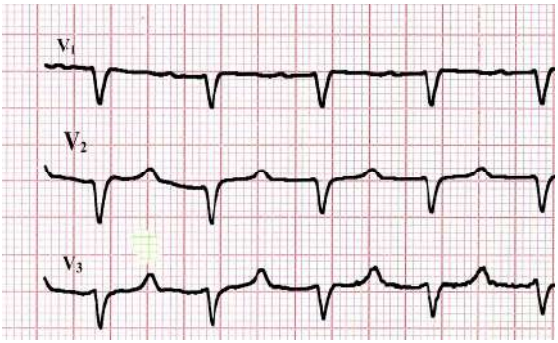


Fig. 6-8. Old MI : Notice Q waves, Isoelectric ST segments, and normal T wave.

Myocardial infarction may be present as :

A) Tall R wave, ST depression upright T wave in V_1 to V_3 posterior MI.
B) New onset LBBB.
C) Poor progression of R wave.

Ventricular aneurysm

To establish the diagnosis, old ECGs are useful for comparison. The absence of ST segment elevation does not exclude the aneurysm.

The diagnostic ECG features are:

- Persistent ST elevation > 4 weeks with ECG signs of old MI (Fig. 6-9) as follows:
  - Deep Q waves.
  - Poor R wave progression.
  - Normal to inverted T wave.
  - Absence of reciprocal changes.

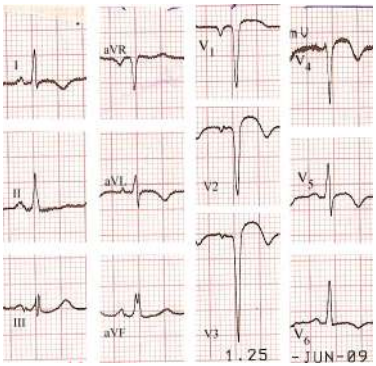


Fig. 6-9. Ventricular aneurysm 6 months following an anterior MI. Persistent ST elevation in V_1- V_5.

C. Site of infarction

The ECG features of MI may be localized in one or more of the cardiac regions (table 6-3):

Table 6-3. ECG localization of MI

Infarction location	ECG leads changes
Inferior MI	**II, III, aVF**
Septal MI	**V_1, V_2**
Anterior MI	**V_3, V_4**
Anteroseptal MI	**V_1-V_3**
Extensive anterior MI	**V_1-V_6, lead I and, aVL**
Lateral MI	**I, aVL, V_5-V_6**
High lateral MI	**I, aVL**
Posterior MI	**Tall R wave and ST depression with upright T wave in V_1- V_2 (reciprocal changes)**
Right ventricular infarction	**ST-elevation in V_{3R}, V_{4R}. Exclusively associated with inferior MI.**

Anatomical localization of MI

The localization of MI according to anatomical regions of the heart as the following:

1) Left ventricular infarctions.
2) Anterior, lateral, inferior, and posterior.
3) Right ventricular infarction.
4) Atrial infarction.

1. left ventricular infarctions

The ECG changes of MI may be localized in the following principal regions of the LV:

A. Anterior myocardial infarction

Infarction of the anterior wall of the LV reflected by ECG changes in leads V_3-V_4 (Fig. 6-10A).

If eight or more leads are involved, it is called extensive anterior MI (see Fig. 6-11).

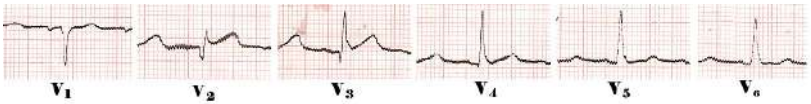


Fig. 6-10 A. Anterior MI, notice ST elevation in V_2-V_4 .

B. Lateral myocardial infarction

Infarction of the lateral wall of the LV reflected by ECG changes in leads I, aVL, V_5, and V_6 (Fig. 6-10B).

If changes are restricted to leads I, and aVL, it is called high lateral MI.

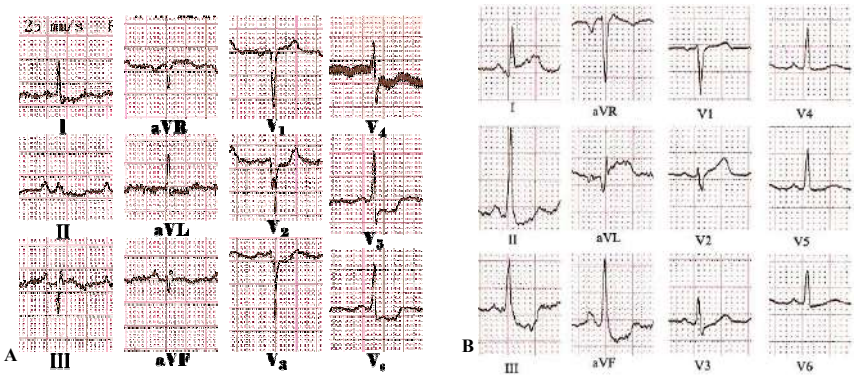


Fig. 6-10 B.

A) Lateral non ST segment elevation MI, notice ST depression in V_4- V_6, I, and aVL.
B) High Lateral myocardial infarction, notice ST elevation in Lead I and aVL, with reciprocal changes in the inferior leads.

C. Inferior myocardial infarction

Infarction of the inferior wall of the LV reflected by ECG changes in leads II, III, and aVF (Fig. 6-10C).

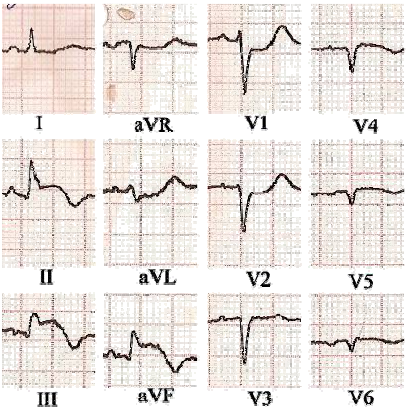


Fig. 6-10 C. Inferior MI : Notice ST segment elevation in II, III, and aVF, also notice the loss of the R wave progression which is replaced by QS in V_2 –V_6 indicates old MI

D. Posterior myocardial infarction

Infarction of the posterior wall of the LV reflected by reciprocal changes in V_1-V_3 (tall R wave, ST segment depression and upright T wave) (Fig. 6-10D).

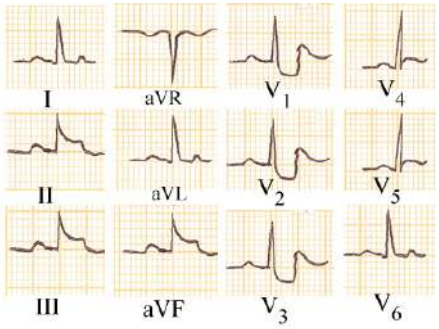


Fig. 6-10 D. Posterior MI, notice the prominent R wave, ST segment depression, and upright T wave in leads V_1, V_2, and V_3, which indicates acute posterior injury. Accompanying inferior MI. the absence of deep S wave in V_4, V_5, and V_6 excludes the possibility of RVH or RBBB as a cause of prominent R wave in lead V_1.

- To confirm the diagnosis of acute posterior MI, additional posterior leads V_7, V_8 and V_9 are useful, which record ST segment elevation (see Fig. 1-13C).
- Commonly, it is associated with inferior MI or sometimes with lateral wall MI.

Practically it is helpful to turn ECG paper upside-down and look at from the back, the ECG changes of posterior MI (Q wave, ST elevation and T wave inversion in Leads $V_1 - V_3$) will appear.

Multiple infarctions

ECG manifestations of two or more myocardial infarction in two or more different cardiac regions. In this situation, exclude the pericarditis if the diagnosis of MI is based on ST elevation alone in more than one discrete region in the heart.

Because overlapping in vascular supply, the infarctions may coexist together as follows:

- **Antero-lateral myocardial infarction**

  Infarction of the antero-lateral wall of the LV reflected by ECG changes in leads V_1 - V_6, lead I, and aVL (Fig. 6-11).

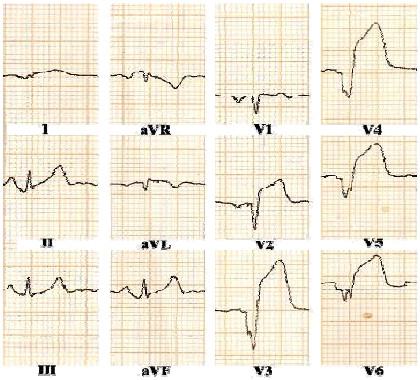


Fig. 6-11. Anterolateral myocardial infarction. Notice is ST segment elevation across the precordial leads V_1 - V_6 ,I and aVL.

- **Antero-septal myocardial infarction**

  Infarction of the antero-septal wall of the LV reflected by ECG changes in leads V_1 - V_3 (Fig. 6-12A).

  Old antero-septal MI has characteristic QS complex in leads V_1 – V_3 with the absence of R wave (Fig. 6-12B), which should be differentiated from other causes of QS complex such as:

  LBBB, LVH, Cardiomyopathy, and LV aneurysm.

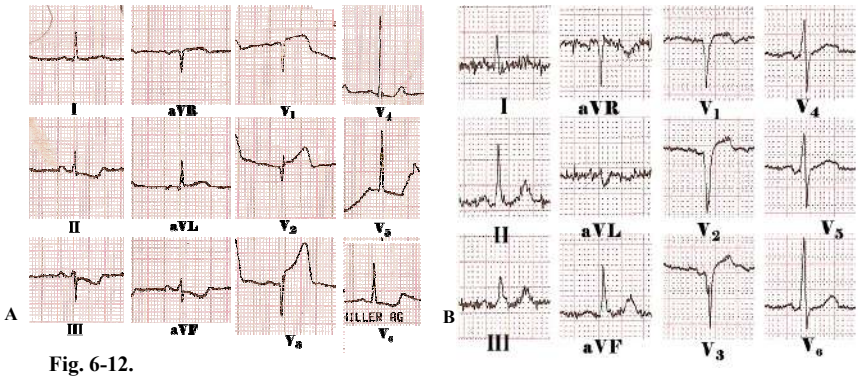


Fig. 6-12.
A- Anteroseptal MI, notice ST segment elevation in lead V_1 - V_3.
B- Old MI, notice QS complex with the absence of R wave in V_1 - V_3.

- **Infero-lateral myocardial infarction**

  Infarction of the infero-lateral wall of the LV reflected by ECG changes in leads II, III, aVF, V_5, V_6, lead I and aVL (Fig. 6-13).

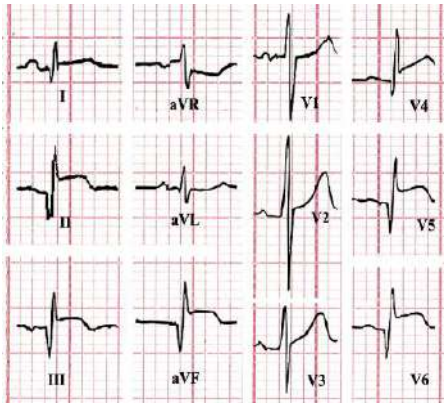


Fig. 6-13. Acute infero-lateral MI, notice ST elevation and Q wave in leads II, III, aVF, and V_4-V_6 .

- **Infero-posterior myocardial infarction**

  Infarction of the Infero-posterior wall of the LV reflected by ECG changes in leads II, III, and aVF and leads V_1–V_3 (see Fig. 6-10D).

2. Right ventricular infarction (RVI)

A | To make the diagnosis of RVI, the right sided chest leads should be recorded (see Figs. 1-13B), looking for ST segment elevation specially V_4R which offers the highest specificity in diagnosis of RVI (Figs. 6-14).

B | Right ventricular infarction, is suspected in the clinical setting of acute inferior wall MI (Figs. 6-14).

C | In the setting of inferior MI, ST elevation in lead III more than lead II, and/or ST segment elevation in lead V_1 increase the possibility of RVI (the absence of these changes does not exclude the associated RVI).

D | ST elevation in V_1 with ST depression in V_2, (a discordant relationship) also suggests the presence of right ventricular infarction.

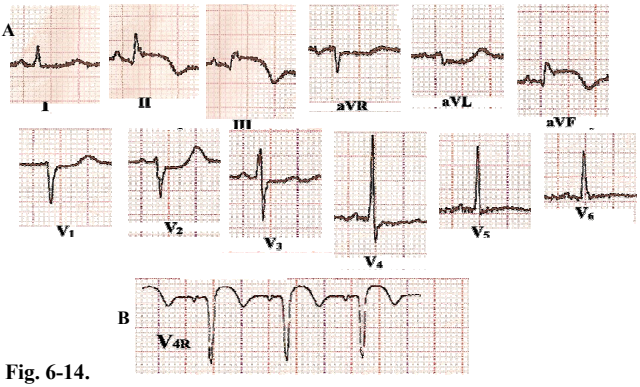


Fig. 6-14.
A- Acute inferior MI with RVI. Notice ST segment elevation and T wave inversion in lead II, III, and aVF indicate acute inferior wall MI.
B- The same patient, the right chest lead V_4R shows ST elevation which indicates RVI.

Practical guideline:

Recognition of the Right ventricular infarction is of major clinical importance, because in patient who is hypotensive with low capillary wedge pressure, the treatment include IV fluid and infusion of dopamine. Vasodilator drugs and nitrate must be avoided.

3. Atrial infarction

- Rarely seen isolated in clinical practice.
- Present with PR segment changes (elevation or depression), P wave changes and abnormal atrial rhythm in clinical setting of other ventricular infarctions (Fig. 6-15).

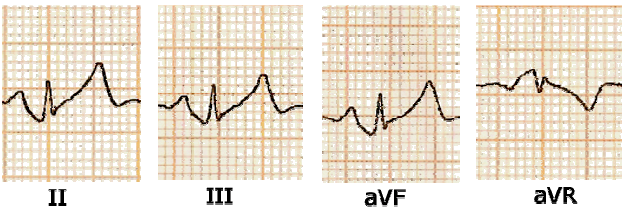
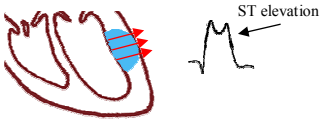
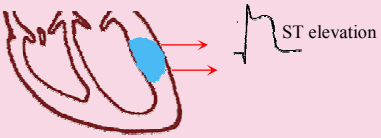
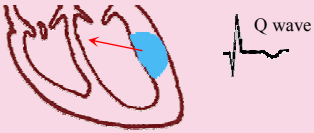


Fig. 6-15. The minimally depressed PR segments in leads II, III, and aVF and PR segment elevation in aVR suggest possible atrial infarction.

The ECG changes, timing, and pathophysiology of ischemic heart disease are summarized in table 6-3.

Table 6-3. Summary of the ECG Changes of ischemic Heart Disease

Diagnosis	ECG changes	Time of ECG changes	Pathophysiology
Stable angina **(exertional chest pain).** **Ischemic ECG changes in the absence of chest pain is called silent ischemia.**	ST depression is the most common change. T wave inversion. T wave pseudo-normalization.	Coincidental with a chest pain or during exercise test.	- Transient, reversible subendocardial ischemia due to fixed stenotic artery, demands supply angina. - With acute subendocardial ischemia the electrical forces are deviated toward the inner layer of the heart causing ST depression or T inversion in leads which face the ischemic area see fig. above.
Coronary artery spasm (Prinzmetal's or variant angina) **Chest pain often occurs transient at rest or night**	Reversible, transient ST segment elevation.	Coincidental with chest pain.	- Spasm may occur in a normal artery or at the site of plaque; usually total occlusion, short duration (minutes); transient transmural (full thickness) ischemia. - With acute transmural ischemia electrical forces (arrows) are deviated towards the outer layer of the heart causing ST elevation in the overlying leads. 
Unstable angina and Non-ST segment elevation MI	ST depression T wave inversion T wave inversion may return to normal or may be permanent.	During chest pain.	- Stenosed artery, but with some blood flow; subendocardial ischemia, then necrosis. - Same mechanism as that of stable angina pectoris but with progressive changes.

Diagnosis	ECG changes	Time of ECG changes	Pathophysiology
ST segment elevation MI **Total arterial occlusion, full thickness ischemia, injury and then necrosis.**	• Tall upright T wave. • Elevated ST segment.	Minutes afterwards	-When transmural ischemia is persistent causing more injury to cardiac muscle, the electrical forces are deviated towards the outer layer causing ST elevation in the overlying leads and reciprocal changes in the opposite leads. -With progressive ischemia, injury, and necrosis, the conduction of electrical forces is decreased resulting in decrease of R wave progression in the overlying leads with small Q wave.
	• Progressive loss of R wave. • Terminal T inversion. • Developing Q wave.	Hours afterwards	
	• ST segment returns to normal. • Deep Q wave. • T wave inversion.	Days afterwards	-Lastly when myocardial necrosis is established, the mean electrical forces are oriented in the opposite direction, so the electrode overlying the area of necrosis records a negative deflection(Q wave)
	• Persistent deep Q wave. T wave inversion may become less marked or may be permanent or may return to normal.	Weeks afterwards	

Practical guideline:

In patient with ECG manifestation of myocardial ischemia/infarction, the other ECG abnormalities should be recognized. e.g., abnormal rhythm, conduction abnormalities because of its prognostic significance.

Interpretation of ST-T changes

Interpretation of ST-T changes is one of the most important areas of clinical ECG in daily practice. While considering deviation of ST segment, J point should be identified (the point of inflection between S wave and ST segment) (Fig. 6-16A). The ST segment should also be identified which extends from the J point to the beginning of the T wave.

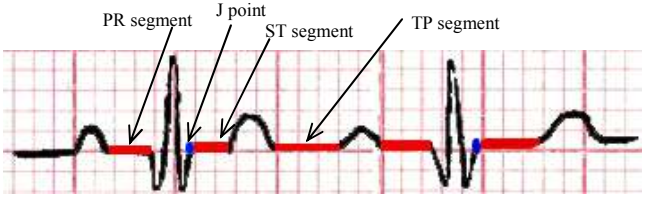


Fig. 6-16 A. Drawing shows the PR segment, J point, ST segment, and TP segment

The total QRS amplitude should be kept in mind, because it affects the amplitude of ST-T changes.

Elevation of ST segment is commonly convex in ischemic heart disease and should be measured at J point in relation to PR and TP segments (Fig. 6-16B).

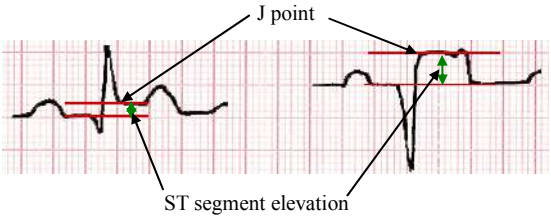


Fig. 6-16 B. Drawing shows the measurement of ST segment elevation at J point in relation to the PR and TP segments.

Depression of ST segment may be horizontal, down sloping, or up sloping (rapidly or slowly) at J point in relation to PR and TP segments (Fig. 6-16C).

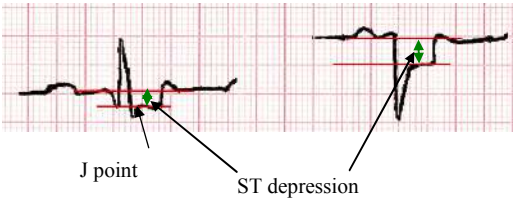


Fig. 6-16 C. Drawing shows the measurement of ST segment depression at J point in relation to the PR and TP segments.

T wave changes should be interpreted in relation to ST segment or in isolation. In ischemic heart disease, it is deep and symmetrical at the beginning and at the end of T wave.

Non-specific ST-T changes:

Minor ST segment change <0.5 mm, T wave inversion < 1mm or ST elevation/depression at the J point itself may be difficult to interpret in clinical practice (Fig. 6-17A).

Fig. 6-17 A. Drawing shows pseudo elevation and depression of ST segment, but the ST segment at the J point form the first shoulder of the T wave, not true ST segment.

The ST-T changes are further divided into primary and secondary.

Primary ST-T changes with normal QRS complex, e.g. Ischemia (Fig. 6-17 B).

Fig. 6-17 B. Drawing shows the maximum of the inversion between the beginning and the end of the T wave, compared to the secondary T wave below the inversion occurs at the end of the T wave

Secondary ST-T changes are associated with changes in QRS complex, e.g. LBBB, LVH (Fig. 6-17C).

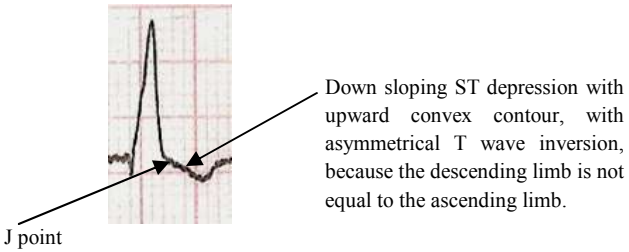


Fig. 6-17C. Drawing shows secondary ST changes.

Memory T wave deep T wave inversion that persists after returning of QRS complex to normal, seen in complete LBBB, RV pacing, wide QRS tachycardia (Fig. 6-17D).

Fig. 6-17D. The first two beats have wide QRS complex with secondary ST-T changes (long arrows), the third beat is sinus. Despite the normal QRS complex, the T wave still inverted, (memory T wave inversion) (short arrow).

Diagnosis of Myocardial Infarction in the presence of Bundle Branch Block

The development of bundle branch block in the setting of myocardial infarction indicates more adverse prognosis.

1. Right bundle branch block (RBBB)

RBBB without MI (see chap. 5)

In RBBB, activation of the septum and LV is the same as normal (Fig. 6-18A), so the initial period of QRS is not affected. Because the right bundle branch is blocked, the RV is activated slowly from the left resulting in new high amplitude R wave in V_1 and deep slurred negative S wave in lead V_6.

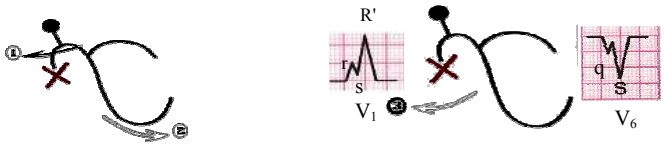


Fig. 6-18 A. Reveals the steps of RBBB as follows:

Arrow 1: septal depolarization from left to right side.

Arrow 2: depolarization of the left ventricle.

Arrow 3: Delayed activation of the RV from the left causes the last part of the wide QRS complex to be positive (R' wave) in lead V_1 and negative (S wave) in leads V_5 and V_6.

Diagnosis of MI in the presence of RBBB

- Generally, the RBBB does not interfere with the diagnosis of MI (ST-T changes in acute infarction and Q wave in old infarction).
- A little bit confusion occurs with antero-septal and posterior MI in setting of RBBB, as follows:

  Antero-septal MI, the necrosis of the septum affects the early depolarization of the ventricles resulting in Q wave and disappearance of initial r wave in V_1-V_3.

  - Acute antero-septal MI: Q wave, ST segment elevation, and T wave changes in V_1-V_3(Fig. 6-18B).
  - Old antero-septal MI: manifested by the presence of Q wave (QR) and absence of initial r wave in V_1-V_3. In this instance, the diagnosis of RBBB is confirmed by the presence of deep slurred s wave in V_5 and V_6.

  Posterior MI

  - Tall R wave, ST depression and upright T wave
  - Commonly associated with inferior MI

  Remember clinical presentation, cardiac biomarkers and previous ECGs are helpful.

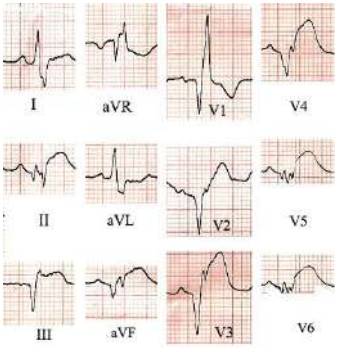


Fig. 6-18 B. RBBB in a patient with acute inferior, lateral and anteroseptal myocardial infarction. Inferior MI manifested by Q wave and ST segment elevation in inferior leads, associated acute anteroseptal and lateral myocardial infarction manifested by Q waves and ST segment elevations in $V_1 - V_6$.

2. Left Bundle Branch Block (LBBB)

LBBB without MI (See also chapter 5)

1) The septum is depolarized from right to left produces q in V_1 and r in V_6 (i.e. loss of normal septal r in V_1 and septal q in V_6) (Fig. 6-19A).

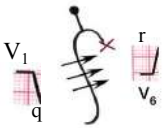


Fig. 6-19 A. LBBB, notice activation of the septum from the right to the left (arrows).

2) In LBBB, the total time for left ventricular depolarization is prolonged. The QRS is abnormally wide, in V_1 wide negative QS complex and wide notched R wave without an initial q wave in V_6 (Fig. 6-19B).

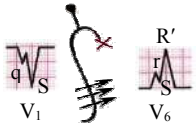


Fig. 6-19 B. LBBB, notice the delayed activation of the LV from the right (arrows), resulting in wide QRS complex with the absence of septal q wave in V_6.

Diagnosis of MI in the presence of LBBB

The diagnosis of MI in the setting of complete LBBB is difficult in clinical practice, but the clinical judgment, biomarkers and the following ECG changes are helpful in diagnosis.

Acute MI (Fig. 6-20C)

- ST segment elevation ≥ 1 mm concordant (in the same direction) with QRS complex in leads V_4, V_5, V_6, lead I, and aVL.
- ST segment elevation ≥ 5 mm discordant (in the opposite direction) with QRS complex in V_1-V_3
- ST segment depression ≥ 1 mm in V_1-V_3

Old MI

The presence of pathological Q wave in the left precordial leads is suggestive of old MI.

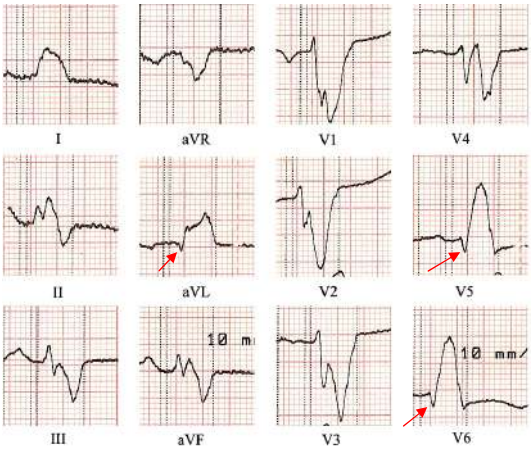


Fig. 6-20 C. Left bundle branch block with lateral MI. The q wave in lead V_5, V_6 and aVL (arrows). Notice the LAD and LBBB indicate poor prognosis.

Practical guidelines:

- *As a general rule, in patients with ischemic chest pain, including those patients with cardiovascular risk factors (as hypertension etc), ECG finding of LBBB is a myocardial ischemia until proven otherwise and cardiac biomarkers should be obtained to confirm the diagnosis. Since the diagnosis of MI in the presence of LBBB is generally masked, or may the LBBB suggests false diagnosis of MI, so the clinical judgment is important.*
- *Three strategies that may help in the correct ECG interpretation of LBBB with MI as follows:*

  - Serial ECGs demonstrating ischemic changes.
  - Comparison with previous ECG.
  - Knowledge of the expected ST segment and T wave morphologies in uncomplicated LBBB is possible, thus, ischemia can be recognized.

- *In early stage of acute MI, the clinical history and ECG are the most important regarding reperfusion therapy, while the biomarkers in this regard are unhelpful, because they are retrospective and time dependent.*
- *Loss of R wave progression in precordial leads or ST elevation in those leads are not enough for the diagnosis of the MI with LBBB.*

CHAPTER 7

Miscellaneous ECG Changes

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Miscellaneous ECG changes

The normal electrocardiogram can be changed by various conditions that are external to the heart; these conditions include therapeutic or toxic level of certain drugs, temperature, endocrine and metabolic abnormalities or interposition of fluid and tissue between the heart and ECG electrodes.

Understanding of ECG changes of these conditions is important, because they are life threatening and treatment should be directed towards the underling cause (anti-arrhythmic and DC shock must be avoided). ECG changes of some of these conditions are discussed in the following chapter.

1-Drugs

- **Digoxin**

  A | Therapeutic effect (Therapeutic effect does not imply digitalis toxicity) (Fig.7-1 A).

  - ST segment depression (reversed tick).
  - Reduction in the T wave size.
  - Shortening of the QT interval.

Fig.7-1A. 'reverse tick' ST segment depression

  B | Toxic effect

  - T wave inversion (change in the T wave during digitalis administration are often the earliest signs of digitalis toxicity).

- Any type of known arrhythmia especially ventricular ectopy.
- Digitalis does not affect the QRS complexes because digitalis does not cause bundle branch block.

Appearance of ectopic rhythm in the course of digitalis administration is nearly always a sign of toxicity until proven otherwise.

- **Quinidine and related drugs** like Procainamide, Disopyramide, Phenothiazine, and Tricyclic antidepressant (Figs. 7-1B).

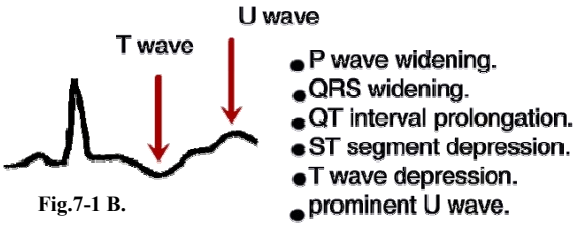


Fig.7-1 B.

Amiodarone is the drug that typically prolongs the QT interval even at therapeutic doses.

2- Electrolytes

A | Hyperkalemia

The earliest ECG evidence usually appears in the T waves. The variety of the changes include:

1) Mild to moderate hyperkalemia (5-7 mEq/L) (Fig.7-2A).

- Tall symmetrical peaked "tent" T waves with narrow base (narrow base T wave may help to differentiate the peaking T wave of the hyperkalemia from other causes like hyper-acute T wave in the earliest stage of acute myocardial infarction).

2) Severe hyperkalemia (8-11 mEq/L)
   - Widening of the QRS complexes.
   - PR segment is prolonged (i.e. delay of the AV conduction).
   - In severe cases, the ECG resembles a sine wave.
   - P wave disappearance (a trial arrest).

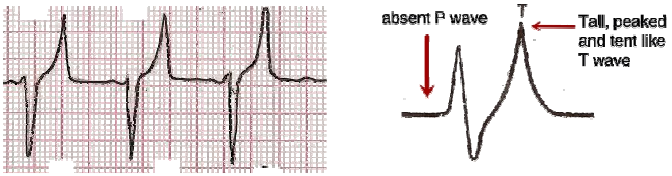


Fig.7-2A. Hyperkalemia, notice the hyper-acute T wave with narrow base.

B | **Hypokalaemia**

- Mild to moderate hypokalaemia (2.5-3.5 mEq/L) (Fig.7-2B).
- Progressive ST Segment depression.
- Progressive decrease in T wave amplitude.
- Increase U wave amplitude.

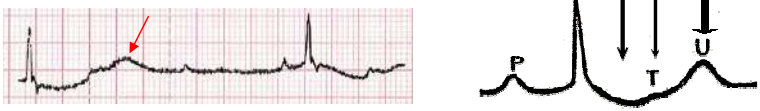


Fig. 7-2 B. Hypokalaemia, notice U wave (arrow).

Sever hypokalaemia (<2.5 mEq/L).

1) Fusion of T and U waves.
2) Increase QRS duration and amplitude.
3) Increase P wave duration and amplitude.
4) QT interval is usually slightly prolonged.
5) Premature beats and sustained tachyarrhythmia.

C | **Hypercalcaemia**

- Marked shortening of the QT interval due to shortening of the ST segment (Fig.7-3A).

D | **Hypocalcaemia**

- Hypocalcaemia is the only condition that can prolong the ST segment without affecting the T wave (Fig.7-3 B and C).

Note the duration of the QT interval is inversely proportional to the serum calcium level.

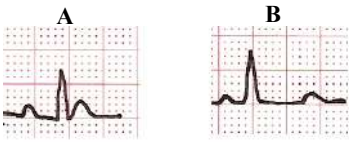


Fig.7-3.
A- ECG changes in hypercalcemia, shortening of the QT interval primarily by shortening of the ST segment.
B- Hypocalcaemia, lengthening of the QT interval primarily by lengthening of the ST segment.

Fig. 7-3 C. Hypocalcemia, notice prolonged QT interval.

3- Renal Failure

The triad of LVH (caused by hypertension), peaked T waves (caused by hyperkalemia) and prolonged QT interval (caused by hypocalcemia) should suggest renal failure.

4- Acute pericarditis

Acute pericarditis may be manifested as follows:

- Diffuse ST segment elevation with characteristic chest pain (Fig.7-4).
- Acute pericarditis almost invariably associated with sinus tachycardia. This is a potential differentiating feature from acute myocardial infarction, which may be associated with either sinus tachycardia or sinus bradycardia.
- Other features that may help in differentiation of ST elevation of pericarditis from that caused by ischemia are summarized in the table 7-1.

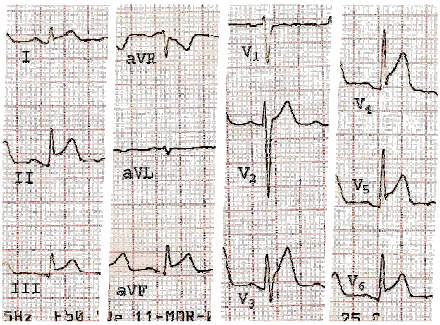
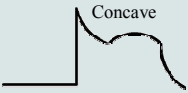
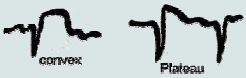
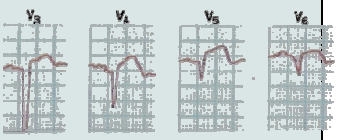


Fig. 7-4. Pericarditis
Key point:
Widespread saddle-shape ST segment elevation.

Table 7-1. ST segment elevation: pericarditis versus ischemia

		Pericarditis	Myocardial ischemia
1	Leads distribution	In most of the ECG leads (diffuse distribution).	Reflect the area, which is affected by the concerned coronary artery.
2	Reciprocal ST depression	Absent, apart from ST segment depression in aVR.	Usually present.
3	ST segment shape	Upwardly concave 	Usually upwardly convex, but may be concave or plateau shaped. 
4	Q wave	Absent	Usually present.
5	PR depression	Often present, although it is specific but less sensitive, (rarely seen).	Absent.
6	Timing of T inversion	T waves inversion, occurs only after ST segment becomes isoelectric.	T wave invert while ST segment still elevated.
	Example ECG strip		

5- Pericardial effusion

ECG changes that are usually seen in pericardial effusion (Fig. 7-5A):

A | Low voltage QRS complexes[1].
B | Low to inverted T waves in most leads.
C | Total electrical alternans.
D | Relative sinus tachycardia.

Fig. 7-5 A. QRS electrical alternans. Note the alternation in QRS height in every other beat in this patient with large pericardial effusion.

Differential diagnosis of low voltage QRS complexes

1) Artifacts as unrecognized standardization of ECG at one half usual gain (i.e., 0.5 mV = 5 mm).
2) Normal variants (may also be seen in pregnancy and obesity).
3) Pericardial tamponade (usually with sinus tachycardia).
4) Pleural effusion.
5) Chronic obstructive pulmonary disease (COPD).
6) Extensive myocardial infarction.
7) Myxoedema (usually with sinus bradycardia "low and slow").
8) Cardiac infiltration (especially cardiac amyloid) (Fig. 7-13).
9) Cardiomyopathy (dilation, usually with diffuse fibrosis).
10) Left pneumothorax (mid left chest leads).

[1] Low voltage QRS complex with reference to the calibration or standardization, which should be set at 1 mV= 10 mm. is defined as:

- The amplitude of the entire QRS complex (R +S) in each of the limb leads is 5 mm or less.
- The amplitude in each of precordial chest leads (V_1-V_6) is 10 mm or less.

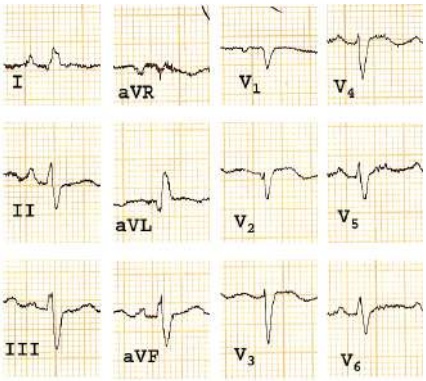


Fig. 7-5 B. Low voltage ECG.

6- Electrical alternans

It is a beat-to-beat variation in the P, QRS and T axis; it can be partial or total.

Partial electrical alternans alternation in the amplitude of R waves (depolarization alternans), this may occur in SVT (Fig. 4-22B), or alternation in amplitude of T wave (repolarization alternans). It is a sign of electrical instability and may precede ventricular tachyarrhythmias (Fig. 7-6).

Total electrical alternans alternation in the amplitude of all ECG waveforms (P, QRS and T). It is usually associated with sinus tachycardia. It is a characteristic feature in pericardial effusion (Fig. 7-5A).

Fig. 7-6. Alternation of T waves amplitude (arrows) without QRS alternans. The clinical significance of T wave alternans indicates high risk of acute polymorphic ventricular tachycardia.

7- Acute myocarditis

The acute myocarditis may be manifested as:

- Non-specific ST-T changes.
- Acute myocarditis should be suspected in patient with unexplained tachycardia, arrhythmia or heart failure.

8- Early repolarization syndrome (high take-off ST segment)

The early repolarization syndrome is a common normal ECG variant in healthy young adults.

The syndrome has the following ECG features (Fig. 7-7)**:**

1) Characteristically, the ST elevation is described as follows:
   A | It may rise 2 to 3 mm above the baseline.
   B | Elevation is stable over an extended period and does not undergo the rapid changes as in pericarditis or MI.
   C | It always follows S wave.
2) Tall R waves and ST-T changes are almost always found in the left precordial leads.
3) Relatively tall T wave, usually more than ST elevation (i.e., ST/T ratio less than 0.25%).
4) No reciprocal changes except ST segment depression in lead aVR.
5) Relatively slow heart rate in comparison with pericarditis, which is commonly associated with tachycardia.

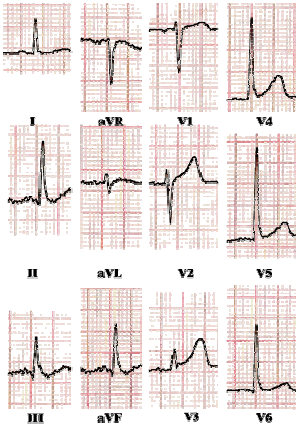


Fig. 7-7. "Early repolarization" normal variant. Early repolarization has to be differentiated from acute pericarditis.

Note:
1. A little barb at the J point, that with ST-T, produces a "fishhook" in several leads.
2. In V_6 the degree of ST elevation is usually less than 25 percent the amplitude of T wave.

Practical guideline:

In general practice, ST elevation is mostly attributed to any of the following causes:

Early repolarization, ST elevation MI or pericarditis. However, it is important to know that in practice, it is often difficult to differentiate between these causes. Thus taking clinical history, comparison with old ECGs and obtaining serial ECG tracing would be necessary to identify the cause of ST segment elevation.

9- ECG changes in Cerebrovascular accidents (CVA)

Usually, ECG changes occur in association with stroke either intracerebral hemorrhage or subarachnoid hemorrhage and others.

These ECG changes occur mainly during ventricular repolarization and are believed to be mediated through the autonomic nervous system and altered sympathetic tone.

The ECG changes of CVA include the following:

1) Abnormally wide T waves that may be deeply inverted or tall and peaked (Fig.7-8).
2) Prominent U waves.
3) Prolonged Q-T interval.

These changes are termed "CVA pattern" and usually resolved with time.

Fig. 7-8. CVA Non-specific change, long QT interval and deep T wave inversion.

10- Chronic obstructive pulmonary disease (COPD)

Chronic obstructive pulmonary diseases with predominant emphysema, affect the heart and consequence ECG as follows:

- Interfere with electrical conduction, resulting in low voltage QRS.
- Pushing the heart down (Fig. 7-9 A) resulting in slow progression of R wave in precordial leads.

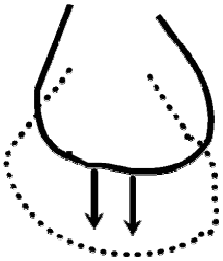


Fig.7-9 A. Diagram shows the position of the heart is lower than the normal position.

11- Pulmonary hypertension

Pulmonary hypertension causes right ventricular hypertrophy and dilatation (Fig. 7-9 B), right atrial enlargement with the following ECG changes:

- Increased P waves amplitude in leads II, III, aVF (P-pulmonale).
- Right axis deviation.
- Occasionally complete or incomplete right bundle branch block.

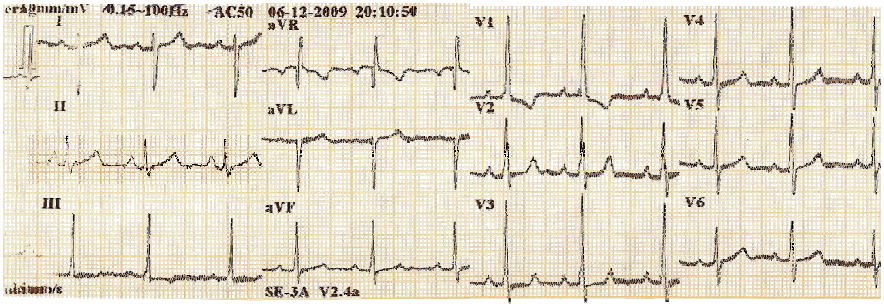


Fig.7-9B. Pulmonary hypertension with right ventricular hypertrophy. Notice the tall R wave (> 7 mm+) in V_1, which is taller than the S wave, a combined voltage of R in V_1 and S in V_6 (>10.5 mm), and the ST depression and T wave inversion in V_1. R wave in lead III is higher than R wave in lead II. P pulmonale (tall, peaked P waves in lead II).

Pulmonary hypertension in the setting of COPD

The combination of COPD and pulmonary hypertension are resulting in the following combination of the ECG findings:

- Increased P waves amplitude in leads II, III, aVF (P-pulmonale).
- Right axis deviation.

- Occasionally complete or incomplete right bundle branch block.
- Low voltage QRS and slow progression of R wave in precordial leads.
- Occasionally SI, SII, SIII syndrome (i.e., S waves in leads I, II and III).
- Abnormal cardiac rhythm commonly multifocal atrial tachycardia.

Notes:

The SI, SII, SIII syndrome may occur in the following circumstances:

- *As normal variants in young healthy adults which probably represent the persistence of physiological dominance of the right ventricular outflow tracts which presents during infancy.*
- *As expression of right ventricular dominance.*
- *Anterior wall myocardial infarction: SI, SII, SIII syndrome may be associated with anterior wall MI especially apical wall infarction. These manifestations tend to be permanent.*
- *The straight back syndrome; is congenital anomaly where the anterior concavity of the upper dorsal vertebral column is lost.*

12- Thyroid abnormalities

There are two main abnormalities:

A | Hypothyroidism, characterized by any of the following changes:

- Low voltage ECG.
- Slow heart rate (sinus bradycardia).
- Inverted T wave without ST segment deviation in some or all leads.

Slow and low ECG indicates hypothyroidism until proven otherwise.

B | **Thyrotoxicosis,** characterized by the following ECG changes:

- Fast heart rate (sinus tachycardia at rest), or unexplained AF.
- High voltage ECG, which may simulate left ventricular hypertrophy.
- Short QT interval.
- A prominent U wave in association with tachycardia should prompt you to think of hyperthyroidism.

13- Obesity

Obesity has the potential for affecting the ECG in several ways

A | Displacement of the heart to the left by elevated diaphragm, but within normal range (electrically horizontal heart).

B | Increasing the distance between the heart and the recording electrodes, although, the true low voltage QRS amplitude rarely appears.

14- Pregnancy

Pregnancy, even at maximal distention (full term) is associated with electrically horizontal heart, but not left axis deviation.

Note:

In full term pregnancy and obesity, the QRS axis deviated leftward but within normal range. The further leftward beyond -30° in obese or pregnant women probably represents a pathological abnormality.

15- Pulmonary embolism (Acute cor-pulmonale)

This may produce any of the following ECG patterns (Fig.7-10):

1) Sinus tachycardia is considered the most common ECG findings.
2) Right ventricular strain, ST-T changes in right precordial leads (V_1-V_2).
3) S_I, Q_{III}, T_{III} (S in lead I, Q in lead III, T inversion in lead III) (Fig.7-10). These changes are more specific but less sensitive and are probably due to acute right ventricular dilatation. They may simulate those produced by acute inferior myocardial infarction.
4) Right axis shift is caused by dilatation and strain of the right ventricle.
5) ST depression results from subendocardial ischemia.
6) Acute right bundle branch block (rSR' pattern in V_1) either complete or incomplete, results from dilatation of the right ventricle.

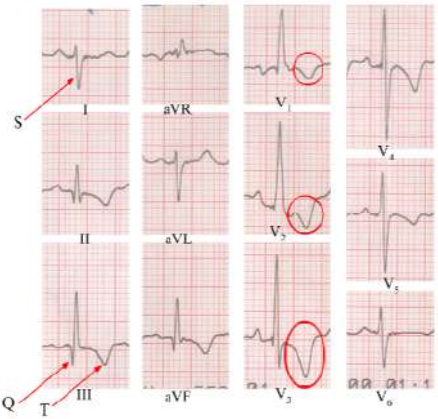


Fig. 7-10. ECG appearance in pulmonary embolism. Notice the characteristic S_1, Q_3, T_3 pattern (Arrows), RBBB morphology and T waves inversion in V_1–V_3 (Circles).

16- Mitral Valve prolapse

The primary MVP syndrome usually has normal ECG, but may be associated with non-specific ECG findings as follows:

1) ST segment depression and T wave inversion in the inferior leads II, III, and aVF is caused by billowing mitral valve leaflets.
2) The isolated T wave negativity syndrome, the T waves are inverted in the midprecordial leads (V_3 and V_4) while the right and left precordial leads reflecting upright T waves.
3) Prolongation of QT interval has reported.
4) Ectopic ventricular beats.
5) Sinoatrial block, LBBB and left anterior hemiblock have been observed in association with mitral valve prolapse.

Notes:

- *The ECG manifestation of mitral valve prolapse is not specific and may vary according to the association with other abnormalities such as accessory pathway. It may also normalize within a short period. Nevertheless, not in all cases with mitral valve prolapse have ECG changes.*
- *Frequent ventricular premature beats and nonspecific ST-T changes, particularly in young patients, should prompt a search for mitral valve prolapse.*

17- Hypothermia

Hypothermia may be associated with the following ECG manifestations:

1) The most characteristic ECG sign is the Junctional (J) wave also known as Osborn wave (Fig.7-11).
2) Wide QRS complexes.
3) Depression of the ST segment.
4) T wave depression.
5) Prolongation of QT interval.
6) Sinus bradycardia.
7) First and second-degree heart block.
8) Ectopic rhythm.

A J wave may be seen in the following conditions:

1) Hypothermia
2) Hypercalcaemia
3) Early repolarization

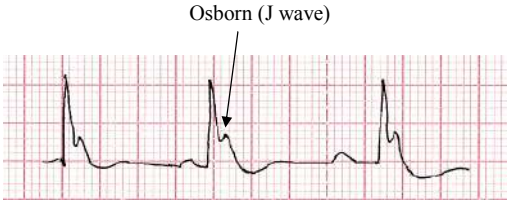


Fig. 7-11. The ECG changes in hypothermia. The Osborn wave (J wave) is frequently seen.

Note:

The ECG manifestation of hypothermia is completely reversible with return to normal body temperature.

18- Amyloidosis

Amyloidosis may be suspected when the following combination ECG changes appear (Fig. 7-12):

1) Low voltage of all waveforms, due to deposition of abnormal protein that interferes with electrical conduction.
2) Marked left axis deviation (the left side is affected more than right).
3) QS or minimal R waves in leads V_1-V_3 or in lead V_4.
4) Prolonged AV conduction time.

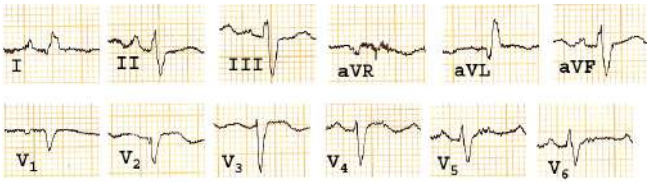


Fig. 7-12. ECG of a patient with heart failure due to Amyloidosis

Note:
patients with amyloidosis are very sensitive to digoxin.

Practical guideline:

It is important to emphasis that, in unexplained wide complex tachycardia, the hyperkalaemia must be considered and treated as early as possible, otherwise a systole and cardiac are the remaining alternative death.

CHAPTER 8

Artificial Cardiac Pacemaker

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Artificial Cardiac Pacemakers (Rapid Review)

The detail of artificial pacemaker lies outside the scope of this book, although a few important aspects of artificial cardiac pacemakers will be mentioned briefly in this chapter.

Key Definitions

1) Artificial Cardiac Pacemaker

Artificial pacemaker is an electronic device that generates electrical impulse, which activates the heart muscle.

A pacemaker system consists of a pulse generator (the pacemaker electronic and power source), and pacemaker catheter electrode (Fig. 8-1).

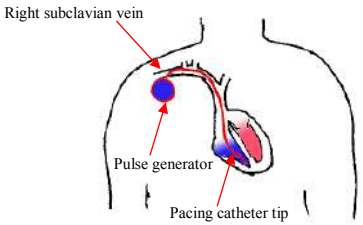


Fig. 8-1. Implanted pacemaker generator (battery) with a wire electrode inserted through the right sub-clavian vein into the right ventricle.

2) Pacing

It is the ability of the pacemaker to send out its own electrical impulse.

3) Capture

It is the response of the heart to a pacemaker impulse that results in atrial and ventricular depolarization.

4) Sensing

It is the ability of the pacemaker to detect the intrinsic electrical activity of the heart. The device is inhibited when the patient's spontaneous rate reaches the preset rate.

5) Escape Interval

It is the time measured from a paced beat to the next normal intrinsic QRS complex (Fig. 8-2).

6) Pacing interval

It is the time interval between two consecutive pacing beats (Fig. 8-2) which is divided into:

A | Refractory period in which a ventricular demand pacemaker does not sense any electrical activity.

B | The alert period follows the refractory period, during which the pacemaker can sense the intrinsic cardiac activity (Fig. 8-2).

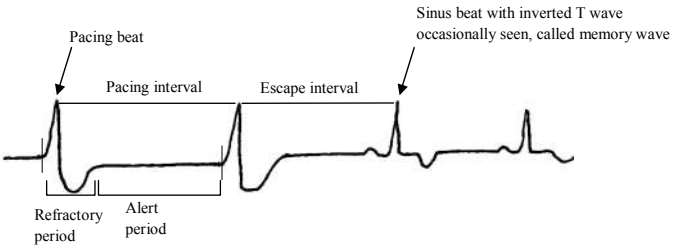


Fig. 8-2. During the refractory period a ventricular demand pacemaker does not sense any electrical activity. The refractory period is followed by the alert period. If no QRS complex is sensed by the end of the period, the pacemaker emits an escape impulse.

7) Demand pacemaker

It is a pacemaker that has ability to work or stop on need, depending on the pre set heart rate.

8) Fixed rate pacemaker

It is a pacemaker, which operates with a fixed rate regardless of intrinsic cardiac activity (rarely used in clinical practice).

ECG findings of normally functioning artificial pacemaker

1) Pacemaker spikes

It is a sharp narrow line recorded on the ECG paper, before P wave when the atria are activated, or before QRS complex when the ventricles are activated, or before both when atria and ventricles are activated (Fig. 8-3).

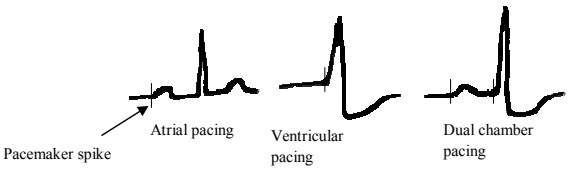


Fig. 8-3. Pacemaker spikes in atrial, ventricular, and Dual chamber pacing.

2) ECG morphology

- Right ventricular pacing (commonly used)

  The ECG shows left bundle branch block pattern, due to delayed depolarization of the left ventricle from the right (Fig. 8-4).

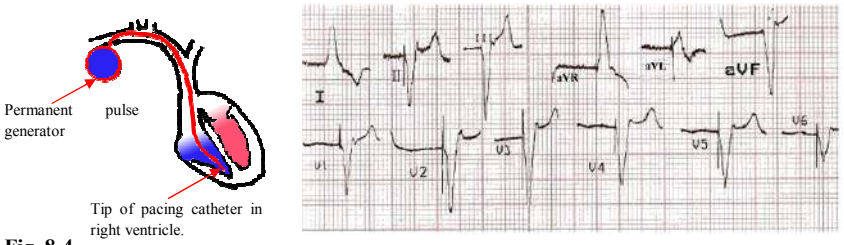


Fig. 8-4.
A pacemaker is inserted in the right ventricle generally produces a pattern resembling that of left bundle branch block, This pattern is caused by delayed depolarization of the left ventricle.

- Left ventricular pacing (epicardial pacing)

  ECG shows RBBB pattern due to delayed activation of the right ventricle from the left (Fig. 8-5B).

- Biventricular pacing this type of pacemaker has a special use for selected cases with heart failure. See p.203

  The ECG shows RBBB morphology with relatively narrow QRS complex (RBBB due to dominance of the left ventricle, and narrow QRS due to contraction of both ventricles simultaneously) (Fig. 8-5C).

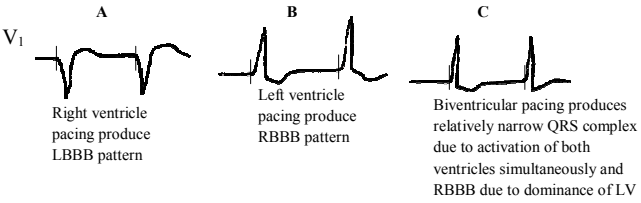


Fig. 8-5 A, B and C. ECG morphology in different types of pacing.

Classification of pacemakers

Generally, pacemakers are classified as:

A | Temporary pacemakers.

B | Permanent pacemakers.

Indications for pacemaker

A | Symptomatic bradycardia

Such as any type of AV block, and Sinus node dysfunction; Symptoms include (syncope, pre syncope, confusion, seizures, or heart failure) and these symptoms must be clearly related to the bradycardia.

B | Asymptomatic bradycardia

Rhythm abnormalities associated with high risk of progression to complete heart block, e.g., Mobitz II AV block.

Pacemaker codes

Five-position pacemaker code is often used to describe pacemaker's function and mode of response, as follows:

- The 1st position indicates the chamber paced.
  (A = atrium, V = ventricle, D = Dual chamber).
- The 2nd position indicates the chamber sensed.
  (A = atrium, V = Ventricle, D = Dual, O = none).

- The 3ed position indicates the pacemaker's response to sensing
  (T= Triggered, I= Inhibited, D= Dual chamber, O= none).
- The 4th position indicates programmable capabilities.
  (R = Rate-responsive).
- The 5th position indicates whether there is a pacing in both atria (bi-atrial pacing) or both ventricles (bi-ventricular pacing).

***In practice**, relatively few modes are actually used, as in the following examples:*

- *VVI, (ventricular pacing, ventricular sensing, inhibited in response to ventricular events).*
- *VVIR, (the same as VVI but also has rate responsivenes[1]).*
- *DDD, (Dual chamber atrial and ventricular pacing and sensing, with dual response either triggered or inhibited to the sensed atrial or ventricular events).*
- *DDDR, (same as DDD but also has rate responsiveness).*

Pacemaker malfunction

The pacemaker malfunction may be as follows:

1) Failure to sense.
2) Failure to pace.
3) Oversensing.
4) Pacemaker-mediated tachycardia.
5) Pacemaker-induced tachycardia.
6) Pacemaker syndrome.

[1] Rate responsiveness (rate-modulated) is means that the pacemaker can speedup when the patient becomes more active and slow down when the patient in rest.

Complications of pacemaker therapy

Pacemaker may produce any of the following complications:

1) Infection.
2) Erosion.
3) Pocket hematoma.
4) Lead displacement.
5) Occasionally the pacemaker also paces the muscles of the chest wall or the diaphragm, resulting in patient's discomfort.

Precautions

- Electromagnetic interference (High-tension cables, Arc-Welders equipment, and High-energy radars).
- Medical equipments and procedures (MRI, therapeutic radiation, electro-surgery, electro-cautery, cardioversion, lithotripsy).
- Digital cellular telephone may cause similar problem but only when the telephone is held in very close proximity to the pulse generator.
- Strenuous or repetitive upper body exercise.
- Works that involve sever shaking.

For a more detailed discussion about artificial pacemakers, refer to Text Book on Cardiology.

Practical guidelines:

- *The major challenge in clinical practice is how to diagnose ischemic heart disease with pacemakers(see BBB with ischemia).*
- *A percussion pacing is performed by delivering gentle blows to the precordium (alongside the lower left sternal edge) to stimulate intrinsic cardiac rhythm, in emergency situation, this technique can be remarkably effective and can get enough time to arrange further treatment appropriately.*
- *Surgeons and anesthetists must always be aware if the patient undergoing surgery has a permanent pacemaker. To avoid interference with, or damage of the pacemaker from dithering, and to minimize the dangers, place the active diathermy electrode at least 15 cm from the box as possible.*
- *Recently the pacemaker has developed for additional specialized uses as cardiac resynchronization therapy (CRT) which pace both ventricles simultaneously (biventricular pacing). It can be used alone or in companion with implantable cardioverter defibrillator (ICD) in selected patients with heart failure.*

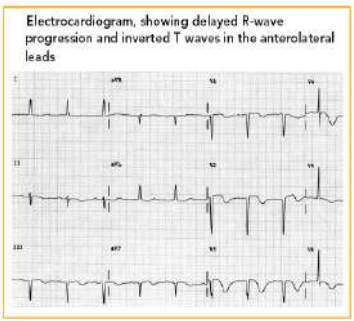
See AHA/ACC2009 updated guidelines for further reading

The following brief discussion is intended to provide only an introduction and rapid review in some recent advance of ECG.

Takotsubo cardiomyopathy

A disorder, or group of disorders, with several names (stress-induced cardiomyopathy, transient LV apical ballooning, Takotsubo cardiomyopathy, and broken heart syndrome) is an uncommon, but increasingly reported cause of acute coronary syndrome. Takotsubo cardiomyopathy is noteworthy for the absence of obstructive coronary artery disease, typical precipitation by intense psychological or emotional stress, and predominant occurrence in postmenopausal women. The characteristic finding of apical LV ballooning is seen on left ventriculography or echocardiography, with transient ST elevation or deep T wave inversion on the surface ECG. Despite the presence of positive cardiac biomarkers and frequent haemodynamic compromise or even cardiogenic shock, almost all patients recover completely, typically with normal wall motion within 1 to 4 weeks.

For further reading see ACC/AHA UA/NSTEMI Guidelines Revision 2009.



Source.

Satoh H, Tateishi H, Uchida T, et al. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hon M, editors. Clinical aspects of myocardial injury: from ischemia to heart failure [in Japanese]. Tokyo: Kagakuyouronsya Co., 1990: 56-64.

QT dispersion

It is the difference between the longest and the shortest QT intervals. The clinical significance is related to the heterogenicity of myocardial repolarization and the consequent cardiac arrhythmias.

Further reading AHA-ACC 2009 guidelines.

Value of lead aVR in diagnosis of acute myocardial infarction

Recently aVR has gained importance in the diagnosis of acute anterior MI. the ST elevation in aVR more than V1 is a marker of left main coronary artery occlusion. Circumflex artery occlusion causes ST segment elevation in aVR but not in lead V1.

For more detail, see the references.

New terminology of myocardial infarction in relation to anatomical location

With reference to electrocardiographic posterior and high lateral MI, new terminology has recently been proposed. These ECG terms "posterior" and "high lateral" MI refers to anatomical "lateral wall MI" and "mid-anterior wall MI" respectively . The impact of these findings and recommendations for standard ECG terminology are still unresolved as of this writing.

AHA-ACC guidelines, circulation 2006; 114:1755-60.

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Testimonials by Reviewers of the First Edition

I have reviewed this book thoroughly, and found it a good effort that provides a well structured text and basic for its subject. Its contents are sequentially presented in a way that it makes it easily understood. It is also comprehensive as far as it tackles all aspects of ECG needed for clinical detection. I would recommend this book as a learning text for medical students and for higher nursing schools as well as for those taking postgraduate studies. My congratulation to Dr. Saleh for achieving this basic standard book.

Dr.Fathi Maklady (MBBCH, DM, FRCP) (UK)
Professor of cardiology
(Suez Canal University) Egypt

I've reviewed this book and found it a newly fashioned one that will add much to scientific creation. Of course, it will give very good help to students in general and postgraduate programmes.

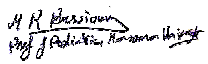
Assoc. Prof. Yahya Alezzy (MD)
(Sana'a University)

With great pleasure, I have undergone reviewing this book of electrocardiography study made for general and graduate students and others interested in internal medicine. It is methodological, orderly structured, and objectively oriented. It helps to understand the principles of ECG and in general practice of internal medicine associated with this field.

Dr. Saeed Alwan
Asst. Prof. Faculty of Medicine
Aden University.

This is an essential work for undergraduate and postgraduate teaching and learning. Pediatric ECG differs from adults. I wish that the author will highlight this side in additional chapter or a separate book later. However, the book covers basic concepts and practical aspects satisfyingly.

Dr.Mohamed Reda Bassiouny (MD, MHPE)
Professor of pediatrics/ Neonatology
(Mansoura University) Egypt



I reviewed this book far-searchingly. Indeed, it shows a great effort done by the author as a one looking for perfection. The book is a good text for internship physicians and postgraduates. It is comprehensive, precise, and appropriate enough as a practical guide.

Dr. Motia Awlaqi
Cardiologist

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